PROBABLE FREQUENCY OF FALSE NEGATIVE CULTURES FROM ROOT CANALS[1]

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A determination of the frequency of false negative cultures from material obtained from root canals, or from the periapical region, has apparently never been reported. The practical significance of such an evaluation is obvious. The culture method is intended to eliminate guesswork. Its accuracy has been taken for granted. The evidence of a culture or of a smear, or both, determines whether we fill the root canals of a tooth. It has often been said, and theoretically at least it is true, that a positive culture is positive evidence that the root canal is not sterile, but that a negative culture is only negative evidence that it is sterile, since infected material may still be present in the root canal and not be shown culturally because of an imperfect technique in obtaining the sample. While the evidence presented in this paper neither proves nor disproves the accuracy of this assumption, it bears upon it indirectly.

Assuming that a negative culture proves sterility of the root canal, the question arises: Is the evidence as presented by one negative culture reliable, or might a culture taken a few days later be positive and thus show the unreliability of a single negative culture? In other words, are we justified in filling a root canal after having obtained a negative culture? A study of twenty-six clinical cases was made in which, after a negative culture had been obtained to determine the bacteriologic status of the root canals or periapical region, another culture was taken immediately before filling the canals. In one of

[1] A preliminary report. Acknowledgment is made to the School of Dentistry of the University of Pennsylvania for the use of laboratory facilities and to the Academy of Stomatology, Philadelphia, Pa., for defraying the cost of materials used in the work. See Proceedings of the International Association for Dental Research: *Journal of Dental Research*, 1933, xiii, p. 206.

TABLE 1

Data showing frequency of false negative cultures from root canals

CASE NO.	NAME	AGE	TOOTH	CLINICAL DIAGNOSIS	DATE WHEN FIRST NEGATIVE WAS OBTAINED	STATUS OF CULTURE	DATE WHEN ROOT CANALS WERE FILLED	STATUS OF CULTURE IMMEDIATELY PRECEDING FILLING OF CANALS
1	P. G.	32	2 u. l.[1]	Chronic ulcerative pulpitis	9–16–29	0	9–18–29	0
2	L. B.	40	3 u. l.	Chronic abscess	2–15–30	0	2–17–30	+
3	E. W.	19	5 l. l.	Pulpitis	6–18–30	0	6–20–30	0
4	L. W.	19	5 l. r.	"Granuloma"	10–22–30	0	10–25–30	0
5	M. H.	17	2 u. r.	Chronic abscess	12– 4–30	0	12– 6–30	0
6	S. G.	33	5 u. l.	Pulpitis	3–11–31	0	3–12–31	0
7	R. O.	25	1 u. l.	Chronic abscess	4–24–31	0	4–27–31	0
8	T. M.	20	5 l. r.	Chronic abscess	4–24–31	0	4–27–31	0
9	E. H.	22	1 u. l.	Chronic abscess	4–30–31	0	5– 2–31	0
10	E. H.	22	2 u. l.	Chronic abscess	4–30–31	0	5– 2–31	0
11	R. F.	13	2 u. l.	Chronic abscess	4–27–31	0	4–30–31	0
12	M. S.	38	5 u. l.	Acute suppurative pulpitis	10–21–31	0	10–26–31	+
13	S. K.	28	2 u. l.	"Granuloma"	12– 1–31	0	12– 3–31	0
14	S. K.	28	3 u. l.	"Granuloma"	12– 1–31	0	12– 3–31	0
15	G. R.	24	1 u. l.	Acute partial suppurative pulpitis	2–19–32	0	2–20–32	0
16	S. R.	22	2 u. r.	"Granuloma"	4–28–32	0	4–30–32	0
17	H. B.	20	1 u. r.	Chronic abscess	5–18–32	0	5–20–32	0
18	M. P.	26	3 u. l.	Chronic abscess	3–31–32	0[2]	5– 9–32	0[2]
19	J. M.	20	6 l. l.	Acute abscess	7–26–32	0	7–27–32	0
20	H. W.	30	2 u. l.	Chronic abscess	12–21–32 and 12–23–32	0	12–27–32	0
21	W. B.	38	2 u. r.	Chronic abscess	12–29–32	0	12–30–32	0
22	R. B.	34	5 l. r.	Chronic abscess	1– 2–33	0	1– 4–33	0
23	W. B.	38	5 u. l.	Chronic abscess	1– 9–33	0	1–12–33	0
24	P. B.	21	6 l. r.	Chronic abscess	2–24–33	0	2–27–33	0
25	J. R.	29	5 u. r.	Chronic abscess	3– 6–33	0	3– 8–33	0
26	D. G.	30	5 l. r.	"Granuloma"	3– 8–33	0	3–10–33	0

[1] u. l. = upper left; u. r. = upper right; l. l. = lower left; l. r. = lower right.

[2] Smear only.

these cases smears were used instead of cultures. These cases represent consecutive trials but not consecutive root-therapy cases.

Cultures were obtained by the following method: When the dressing removed from the root canal presented no obnoxious odor and appeared clean, a smear was made. The dressing was then discarded. If the smear showed no microörganisms, a fresh sterile paper-point was inserted into the canal and a culture taken. In some cases the smear was dispensed with and only the culture method used. For this purpose a fresh paper-point was sterilized in a molten-metal sterilizer, inserted into the root canal as far as the apex, and worked back and forth in the canal for about one minute. When the point was removed, it was wet at the tip for a distance of at least 1 mm. If it was not, a drop of the culture medium was conveyed to the pulp chamber with a sterile instrument, and worked to the apex with a sterile smooth broach for about one minute. A sterile paper-point was then inserted into the root canal for one minute, to absorb the moisture. This method was found to be more satisfactory than an attempt to insert into the root canal a paper-point saturated with culture medium, which procedure usually resulted in buckling or curling of the point. In either case, the point was dropped at once into a tube of culture medium.

The culture medium was principally hormone broth, but occasionally Rosenow glucose-brain broth, or Rosenow liver-broth, was used. The same kind of culture medium was used for the same patient throughout the study. In all these cases an antiseptic dressing [either camphorated chlorphenol, or zinc iodide (*M*) with iodine (0.1 *M*)] was sealed into the canal between the sitting when a culture was taken to determine sterility of the canal, and the following sitting when a check-culture was taken just before filling the canal. In this way an "extra treatment" was ordinarily given the tooth before the canals were filled.

Of the twenty-six cases studied, two showed growth (*table 1*). One of these two cases (No. 12, M.S.) had developed a peridentitis when seen on 10–23–31. But since the culture taken on 10–21–31 had been negative, and the tooth was comfortable on 10–26–31, the canals were filled on that date. No "alibi" is intended in mentioning this, however, because in case No. 18 (M.P.) a longer period of time than usual

elapsed between the last negative smear and filling of the canals, yet the smear taken just prior to filling was negative. In the two cases where positive cultures were obtained before filling the canals (Nos. 2 and 12), no attempt was made to determine whether the growth was due to contamination. On the whole, therefore, the evidence seems to indicate that the probable frequency of false negative cultures is less than 8 percent. This evaluation takes for granted that a sufficient sample for culturing has been removed from the root canal or, preferably, the periapical region.

Conclusion. Following a technique for culturing root canals in which a fresh sterile paper-point was inserted to the apex of the canal (or beyond into the periapical region), so that upon removal its tip was moistened for a distance of at least 1 mm., it was found that the probable frequency of false negative cultures so obtained is less than 8 percent.

CRITIQUE OF A NEW THEORY OF MAMMALIAN DENTAL EVOLUTION

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Study of the classification, relationships, and evolution of mammals, living and fossil, should take into account all their structures and even their physiology and habits, so far as known, but in practice it must rely more on the morphology of the dentition than on any other one sort of evidence. Many efforts have been made to establish a general theory of mammalian dental evolution, and the published theories are almost as numerous as the students who concern themselves with mammal teeth. They range from ignorant fantasies to the elaborate and thoroughly documented results of a lifetime of able and detailed work. A new theory has just been published by Dr. M. Friant in a work issued from the Laboratoire d'Anatomie Comparée of the Muséum National d'Histoire Naturelle, in Paris, as an attractive volume initiating a new series of publications from that historic museum.[1] In a eulogistic preface, the distinguished French mammalogist and comparative anatomist, Dr. R. Anthony, says (p. ix)[2] that "one can foresee that such a work [the systematic application of Friant's theory to all mammals] will lead to a complete reworking of the broadest lines of taxonomy. The publication which I present opens, then, an inexhaustible mine of original investigation to anatomists and zoologists. I consider it as certain that this new conception of the mammalian dentition will find rather rapid acceptance."

A work so sponsored is necessarily important, and such an authoritative prediction of the basic remodeling of current ideas imposes on all

[1] Friant, M. 1933. Contribution a l'étude de la differenciation des dents jugales chez les mammifères. Essai d'une théorie de la dentition. Publications du Muséum National d'Histoire Naturelle, No. 1, pp. i–ix, 1–132, figs. 1–71.

[2] This and other quotations are translated, closely following the sense of the original French.

students the duty of examining the theory and, if it is found acceptable, of starting the herculean task of renovating a whole science. This review proposes to abstract the elements of the Friant theory and to criticize certain of the data, deductions, and hypotheses involved in it. The presentation of the theory is, of course, only a very brief résumé—the original theory should be read—but the critique of it is based on careful study of the whole work (and several preliminary papers by Dr. Friant) and it is believed that it permits proper evaluation of the theory.

Friant gives three possible explanations of the multiplicity of cusps on mammalian teeth: (1) budding of new cusps, (2) concrescence of teeth originally separate, and (3) functional stimulus. So stated, as she recognizes, the third is not really an alternative but might be the causal explanation regardless of the morphological history. She rejects the first two explanations as false. The functional stimulation, or literally "excitation or irritation," is supposed to come from actual movements of the jaws in contact with each other before the teeth are fully formed, hence beginning and particularly important *in utero*, and no longer of any importance when the teeth are in use. The form of the teeth of each individual is determined developmentally by the exact nature of embryonic jaw movements.

The basic types of molars are then classified by Friant as follows: *Bunodont*—a large number of similar cusps separated by longitudinal and transverse valleys of equal depth. This is supposed to arise by jaw movements equally developed in every direction in the horizontal plane. (This use of the term "bunodont" is different from that of any other author.) *Belodont*—cusps arranged in longitudinal rows. This is supposed to result from a predominance of lateral movement over all other movements in the horizontal plane. *Toechodont*—cusps becoming united transversely before they are united longitudinally. This is supposed to result from a predominance of anteroposterior movement. *Abachodont*—a checkerboard pattern very similar to the bunodont but with the valleys generally deeper, supposedly produced by the imposition of jaw movements at right angles to those (transverse or anteroposterior) which were originally most active and had already produced "belodont" or "toechodont" patterns. Friant emphasizes the fact that these ideal patterns are never found in a pure

or clear condition in actual teeth, and further that there are always cusp unions in both longitudinal and transverse directions but that only those which occur first (embryologically) are to be considered in determining the dental type.

Applying this theory to morphological history, Friant says that the complexity of mammalian teeth as opposed to the simplicity of the teeth of lower vertebrates is the direct result of the unique mammalian ability to move the jaw in a horizontal plane and not only vertically. She goes on to say that all Mesozoic mammals fall into two groups, triconodonts and multituberculates, and that of these the triconodonts represent a secondary simplification while the multituberculates represent the primitive type which have "belodont" teeth little advanced beyond her hypothetical "bunodont" condition. From this "bunobelodont" multituberculate dentition, all others were derived by various changes in emphasis on some particular direction of jaw motion in the horizontal plane. Development of carnassial teeth and, eventually, of simple unicuspid teeth, as in some seals, follows emphasis on vertical motion and eventual loss of horizontal motion.

Before examining some of the more concrete considerations on which these views are based, certain outstanding difficulties inherent in the general theory may be mentioned. In the first place, as Friant says (p. 112), "it goes without saying that besides the indubitable purely individual reaction of a tissue to a mechanical action, it is necessary to accept, here as everywhere else, the hypothesis of a fixation of the arrangements acquired in the course of successive generations, permitting the results to accumulate. We do not insist further on this point which is, in sum, the postulate necessary for any attempt at a causal explanation of a characteristic anatomical structure of a zoological group of greater or less extent." But, as is too well known to require more than bare mention, most modern biologists reject this postulate (of the inheritance of acquired characters) as probably false and certainly unproven, and the present consensus is that it is not essential to a causal explanation of structural evolution. Friant's theory, or hypothesis, is not on this account to be rejected without further trial, but a grave doubt is introduced as regards its most basic postulate and the evidence for the theory must be very strong to overcome this fundamental weakness.

On a second point some inherent improbability must be urged against the theory, and the lack of a good factual basis pointed out. There seems to be no good evidence that embryonic jaw movements do, in fact, significantly influence the structure of developing teeth. That some jaw movement is possible in the embryo is clear, and that some movement occasionally does occur is also fairly well established, but that this is constant and characteristic and that it influences the tooth pattern is not proven and is extremely improbable. The evidence opposed to this hypothesis is very strong. For instance, if it is true, how are we to explain the not infrequent occurrence of teeth anomalous in position and so placed that the opposing jaw cannot possibly have influenced their developing germs, which are nevertheless of the definite pattern characteristic for the species? And what reasonable explanation, on Friant's theory, can be given for the fact that posterior premolars, often complex, definitely patterned, and molariform, are throughout their formation shielded by the overlying deciduous teeth from any direct influence of jaw motion? Last premolars and their deciduous predecessors are frequently almost identical in pattern, yet their mechanical environment during development is certainly very different and the pattern would therefore also be very different if this theory were true. Furthermore, the last molar may develop its characteristic shape without any direction or influence from jaw motion, for in many if not in most cases it does not acquire this shape until after other teeth are in use and are preventing any jaw contact in the region of this developing tooth.

A third inherent difficulty in the whole theory lies in its attempt to have an almost unlimited number of different and complex dental forms defined and differentiated by a very limited repertory of simple jaw movements. Friant herself distinguishes only three types of jaw movement, anteroposterior, transverse, and vertical, yet, singly or in combination, these are supposed to have caused all mammalian dental patterns, the number of which probably reaches tens of thousands. This objection is perhaps not fatal: with some risk of a *petitio principii* it is always possible to say that an infinite number of combinations, too subtle for analysis, may be derived from three basic motions. But it certainly is legitimate to doubt whether there are in reality the almost innumerable distinctive and separate types of jaw motion demanded by this theory.

Friant singles out two previous theories for special attack: the tritubercular theory and the premolar-analogy theory. These are not really separate theories, the latter being merely a modern development of the former. The essential, well founded, and really important part of the tritubercular theory (which I prefer to call the Cope-Osborn theory because the name "tritubercular" leads to verbal misunderstanding and tends to obscure this essential point) is the recognition of a primitive stage of molar structure, trigonal or (structurally if not literally) tritubercular in the upper jaw and "tuberculosectorial" in the lower, through which most or all marsupial and placental mammals are supposed to have passed. The premolar-analogy theory does not contradict this; on the contrary, it accepts it and adds new and strong evidence in its favor. In a part of the "tritubercular" theory which Osborn recognized as being much more speculative and not essential to the later history and to the broad value of the whole theory, he also attempted, many years ago, to explain the origin of this primitive type by rotation of terminal cusps in a triconodont molar and subsequent modification of the literally tritubercular molar thus formed. This part of the theory has been rather thoroughly disproven. As far as I know, no one believes in it today, not even its author. It is not essential to the important part of the theory, nor was the latter ever based on the supposition that the triconodont type was ancestral to later types. In attacking this view, Friant is following in the footsteps of all the present adherents of the theory she is opposing, and her own arguments, and many more, have already been so fully discussed by them that further comment on this point is not needed.

It is, however, necessary to consider briefly some of Friant's objections to the belief that the trigonal-tuberculosectorial dentition is primitive. She says (p. 28): "Now, it clearly appears that the forms [of Primates] with quadrate molars of quadricuspid type, like *Necrolemur* (Middle Eocene of Europe) and *Microchoerus* (Upper Eocene of Europe) among the tarsioids, *Adapis* (Upper Eocene of Europe) among the lemuroids, are as ancient as, if not more ancient than, the forms with molars of tritubercular type." This is not an adequate expression of the facts. Many primates are known from the Middle Paleocene to the Lower Eocene, some millions of years older than any mentioned by Friant. These are practically all functionally trituber-

cular. Furthermore, when the structural evolution can be followed in successive stages, as in the *Pelycodus-Notharctus* group, it can be seen clearly and without reasonable doubt that the quadrate type is developed from the trigonal. These observations are not hypothetical or theoretical abstractions. They are based on literally hundreds of specimens of the fossilized teeth of animals that actually lived in successive geological epochs.

Then Friant raises the same doubt regarding condylarths and creodonts, suggesting that quadrate types are as old as trigonal molars. But if the actual fossils are studied in the order of their known ages, this is immediately disproven. The great majority of the early condylarths and creodonts have trigonal upper molars; and when the actual ancestors of those with quadrate molars are known, the fossils clearly show that they were also derived from trigonal types and that the acquisition of quadrate molars is progressive. This is abundantly illustrated by the work of Cope, Osborn, Matthew, Gregory and many others on the Paleocene and Lower Eocene.[3]

The Paleocene mammals are of crucial importance in the development of any reasonable theory of molar evolution and their neglect is a weak point in several recent studies, particularly by authors who have not had occasion or opportunity to study these forms at first hand. The Mesozoic mammals are of still greater importance and any theory worthy of consideration must take them into account, even if not actually using them as basic evidence. Friant recognizes this. She discusses the Mesozoic mammals, reproduces figures of some of them, and gives them a very prominent place, deriving all later dentitions from an essentially Mesozoic type, the multituberculate. But unfortunately Dr. Friant's understanding of Mesozoic mammals is both inadequate and inaccurate.[4] She divides them into two groups,

[3] Friant also errs remarkably in saying (p. 28) that paleontologists agree in deriving perissodactyls and artiodactyls from *Phenacodus* or similar condylarths.

[4] Within the twelve years or so preceding the completion of Friant's study there were published two comprehensive memoirs and about thirty papers exclusively devoted to Mesozoic mammals, with numerous reviews or more incidental references and discussions. Our knowledge of Mesozoic mammals was completely revised and greatly altered during this period. New discoveries of fundamental importance were also made (especially the Cretaceous forms of Mongolia). It is with some diffidence that I call attention to the neglect of work most of which was done by me (with much also by Gregory and some by

multituberculates and triconodonts. Now it has been known for over fifty years that there are at least three major groups, with respect to molar structure, in the Jurassic alone, and that there are four was long since suspected and recently proven. The two of really fundamental importance for the study of molar evolution are completely omitted by Friant. She says (pp. 31–32): "The founders and supporters of tritubercuIy base themselves on the affirmation, very important if one could consider it as indisputable, that the most primitive mammals have tricuspid teeth, that is, with the three cusps in a straight line"; and she lists, as representative of this type, *Dromatherium*, *Amphilestes*, *Triconodon*, and *Dicrocynodon*. As regards this list, *Dromatherium* is not a mammal at all, and "*Dicrocynodon*" (the correct name of which is *Docodon*) is so far from being a simple triconodont that it actually has the most complex molars known in any pre-Cretaceous animal, with lower molars consisting of seven distinct cusps, a dozen ridges and crests, and many grooves, hollows, and basins, all arranged in a very distinctive pattern and by no means in an anteroposterior line. As regards her general statement, as mentioned above, it has been many years since any supporters of the general theory of tritubercuIy, or indeed the founder of this theory, have believed that all the ancestral mammals had triconodont teeth; and they have never believed that all the most primitive mammals had such teeth.

Friant believes that small mammals tend, because they are small, to have simpler teeth than large mammals, and on this basis she explains the simplicity, secondary as she believes, of the triconodont teeth. The multituberculates are, in her view, really primitive and ancestral, while the triconodonts are secondarily simplified because they are so small. Even if this were true it would have no particular bearing on the modern form of the Cope-Osborn theory, but it is not true. By far the largest Jurassic mammal known is a triconodont (*Trioracodon major*) with relatively simple teeth. The triconodonts as a group average larger than the contemporaneous multituberculates. In the Cretaceous the multituberculates also average at least as small

others), but I do this, not from any sense of pique, but only because all of Friant's errors in this field would have been avoided by some knowledge of recent and readily accessible work, of which she makes no mention and apparently is unaware.

as the contemporaneous tritubercular or tuberculosectorial animals (mostly marsupials), and furthermore many of the smallest multituberculates have considerably more complicated molars than do the largest (for example, *Cimolomys*, less than half as large in linear dimensions as *Meniscoëssus* and with over a third more cusps on M_1). Indeed, Friant's view that large animals have more complicated teeth than analogous or related small animals is open to grave doubt. They sometimes have, but there are so many exceptions that this may not even be a real tendency, however slight, and can hardly be a factor of primary importance in molar evolution. Each of Friant's examples could be offset by others exactly opposite. Some are given above and numerous others could be mentioned, almost at random, for example: *Carolozittelia* has more complicated teeth than its much larger descendant *Pyrotherium*. The largest species of *Hypohippus* have much simpler teeth than the majority of the smaller contemporaneous or ancestral horses. Small dasyure molars have more cuspules and are more complicated than those of their considerably larger relative, the thylacine. Most small primates have more molar cusps than their relatively gigantic ally, man. The sperm whale, largest mammal that ever lived, has almost irreducibly simple teeth, and the smallest living mammals, shrews, have rather complex multicuspidate molars. If in some groups there is a tendency for molar complication and increase in size to go more or less together, this appears generally to be because both are independently progressive characters of the group rather than in any exact sense an effect and its cause.

Friant's statements regarding occlusion, jaw motion, and molar evolution in the multituberculates are also incorrect, and this is of prime importance as she singles out the multituberculates as structural ancestors of most or all later mammals. She considers the molar occlusion to be in and between three alternative positions: median, the two lower cusp-rows in the two longitudinal upper valleys; internal, the outer lower cusp-row in the inner upper valley; and external, the inner lower cusp-row in the outer upper valley. In fact the last two positions were impossible in the living animal. In forms with two lower and three upper cusp-rows, the lower rows always and invariably occluded in the two upper valleys. She considers the jaw motion to have been predominantly lateral: this is the fundamental character

of her "belodont" type toward which the multituberculates are supposed to be tending, and it is her explanation of their possessing longitudinal cusp-rows. In fact anteroposterior motion was always predominant and indeed no other was even possible, while the teeth were in contact, perhaps with the partial exception of a few forms (*Taeniolabis*) in which there may have been very slight and unimportant lateral movement, along with the decisively predominant anteroposterior movement. She considers the two-rowed upper teeth of the Jurassic multituberculates to be derived from three-rowed types. In fact this is a reversal of the evidence and of any probable reality, for the known allied three-rowed types are all literally millions of years later[5] and there is a large body of conclusive evidence that they arose from the two-rowed forms.

It is not clear to me whether Friant considers the multituberculates as actual ancestors of later mammals or only as possessing structurally archetypal molars, but if I understand her implications correctly, the former may be the case. This would be still more extraordinary (one might say impossible) than the collocation of rodents and elephants which she explicitly makes. Here appears another point which greatly weakens her dental theory, and, I believe, completely vitiates most of the phylogenetic views expressed, or hinted at as the subject of future publication. She appears to base herself almost entirely on selected individual teeth, taking no account of the anatomy of the other parts of the body or even, in almost all cases, of other parts of the dentition. Only by a much broader point of view and background, where the data are available, can an investigator hope to escape the dangerous pitfalls set for him by the almost universal evolutionary phenomena of convergence and divergence. And this broad background, whether expressed verbally or implicitly demonstrated by the nature of the conclusions drawn, needs also to be accompanied by an exact sense of sequence, as demonstrated by the known ages of all the pertinent fossil dentitions yet discovered.

Friant also attacks the "reigning" theory, this time in its modern or premolar-analogy form, on the ground that progressive simplification is a common evolutionary process and that constant progressive

[5] The three-rowed *Tritylodon* and its allies are earlier, but their relationship to the two-rowed types is certainly not ancestral, probably very distant, and possibly wholly illusory.

complication is not theoretically acceptable. So far as I know, no competent recent student has ever denied that progressive simplification can and does take place, but this does not in any way weaken the "premolar analogy" point of view (which is based on much more evidence than premolar analogy). "It seems rather," Friant adds (p. 46), "that premolars are molars reduced as a result of the place which they occupy in the jaw." That this is an exact reversal of what actually happened in the many instances pertinent to the discussion is not a matter of dispute. We have fossil series, such as that of the horse, which no evolutionary biologist can reasonably deny to be actual progressive structural lines. There are no grounds for dispute or theorizing, for the objective record of the real sequence of structural events lies before us: the premolars begin as simple teeth in the oldest forms and become progressively complicated and eventually quite molariform. That this did not always happen is clear; under either theory there is no reason why it should, but to say that it therefore never happened is flatly contradicted by the actual records of fossil dentitions in our collections. On the other hand, I do not know of any established sequence in time in which the premolars begin by being molariform and end by being simple and primitive (as that word is generally understood).

Friant devotes considerable space to the study of the order in which the different teeth and the different cusps of each tooth are calcified. Observations on the order of cusp calcification and the relative times at which different cusps become fused together are based on the horse, rhinoceros, and a number of rodents. This is her original field of work and Dr. Friant's observations on it are interesting, valuable, and undoubtedly entirely accurate. Perhaps the most interesting, if not the most original point brought out is that rodent teeth with high crowns and deep enamel-lined tubules (e.g. *Hystrix*) are cuspid as originally formed and not fundamentally different from brachyodont types (e.g. *Sciurus*), differing chiefly in the depth at which the various cusps unite and to which the blades and pockets extend. This and other observations in this chapter need no special discussion as they are equally explicable by any current theory of molar evolution and as Friant's theoretical conclusions are discussed adequately in other connections. One point, may, however, be mentioned. In laying

great stress on the general order of cusp union as an index of molar evolution, Friant takes this as a fundamental structural character of each natural group. Other theories accept it as a character of the same status as any other, subject to the same sort of evolutionary history, to divergence, convergence, etc. The paleontological evidence seems to be strongly opposed to Friant's attitude.[6] As an example, I believe that no one will deny that *Protohippus* and *Hipparion* are closely related animals, and that they had a not very remote common ancestry. In their anatomy there is no visible suggestion of any difference in jaw motion. They are contemporary and of about the same size. But in *Hipparion* the protocone does not unite with any other cusp until long after all others are firmly united, while in *Protohippus*, and also in some of the common ancestors of both forms, the protocone unites very early and may in fact unite before some of the other important connections are established (i.e., higher on the crown). This is a decisive example, and many others could be given.

Much might be said of the relationship of jaw motion to fundamental molar types, but a few objections and concrete examples will reveal weakness in the new theory in this respect also. Horizontal motion, its author says, is the distinguishing feature of mammals and the cause of their molar complexity. It has long been recognized that jaw suspension and dental differentiation are related phenomena, but Friant's interpretation of them as direct cause and effect seems to be untenable. There are, as she mentions, early reptiles which have a high degree of cusp development. But, as she does not point out, they still had a reptilian jaw suspension and many of them had not progressed significantly toward the mammalian type in this suspension and the masticatory musculature even though they had done so in dentition. Some (e.g., among diadectids) had molars with transverse rows of cusps which, according to Friant, must be a result of antero-

[6] If paleontological evidence seems unduly stressed in this critique, it should be borne in mind that when the actual sequence in a morphological history is discussed, paleontology must supply the crucial and the only direct evidence. Furthermore, this is in line with Friant's own work, for she also has frequent resort to paleontology and in introducing her, Dr. Anthony says (p. vii): "The author of the work which I present was particularly well equipped to attack the problem . . . . she had always particularly interested herself in the study of fossil forms; she was better informed on paleontology than young zoologists usually are."

posterior motion, yet the jaw suspension is thoroughly reptilian and any considerable degree of such motion is out of the question. As mentioned elsewhere, Friant's theory demands that the multituberculates have predominantly lateral motion, but the fact is that such motion in them was certainly extremely subordinate or totally absent. In polydolopids, also, the relationship of cusp union and jaw motion is opposite to that demanded by the Friant theory. Hyracotheres are supposed to be developed from the "buno-belodont" type, but in fact in them the cusps are not uniting longitudinally but transversely; and the predominant motion was apparently transverse, which is right for a "buno-belodont," on Friant's theory, but which should have produced crests in the opposite direction. But examples might be multiplied indefinitely to show how illusory is this supposed correlation of jaw motion and cusp formation, and hence how impossible it is that the motion can have been the real cause of cusp evolution as postulated in this theory.

Dr. Anthony's introduction (p. ix) contains the significant observation that "furthermore, her theory is rigid, as any good theory should be, in the sense that it is impossible, even on one given point, to substitute for her interpretations other interpretations without bringing down the whole edifice." To the reviewer, it seems not only possible but obligatory to substitute other interpretations for those of the author of this new theory at many different points, and the theory thereby becomes untenable.

This critique may close by quotation of the concluding passage of the work it discusses (Friant, p. 132): "One will then see, I think, that dental characters permit the establishment of unions and of separations which can already be foreseen: that the hyrax and *Arsinoitherium*, to give only these single and characteristic examples, today classified for some unknown reason alongside the proboscideans, enter, on the contrary, into the perissodactyl ungulates in the most natural way; the affinities of the rodents with the proboscideans will also become clearer and more visible. These are the special studies which I plan to pursue, basing myself on the general conclusions which I have tried to set forth here."

IMMUNOLOGICAL REACTIONS IN DENTAL CARIES[1]

G. GRAHAM MACPHEE, M.A., M.B., CH.B., L.D.S.

Pathological and Bacteriological Laboratory, School of Dental Surgery, University of Liverpool, Liverpool, England

1. In 1912, von Beust (1) wrote that it is "possible, even probable, that the immunity to caries . . . is the result of the formation of antibodies." Fish (2), who in 1926 considered it "worth while investigating the possible existence of an active immunity to caries in the blood stream," in 1932 admirably described (3) a process of reaction of the tooth to natural and experimental caries. Immunity to caries may therefore be considered in this light as correlative to that form of immunity to *contagium vivum* utilized *in vitro* for laboratory diagnoses.

2. In a short investigation, commenced in September 1932 at the School of Dental Surgery, University of Liverpool, it has been found that, (a) in four volunteers, the pathogen-selection culture method of Solis-Cohen (4) showed indications of blood-selection of the streptoccal group in two cases of progressive caries, and gave a sterile result in two cases having small non-progressive cavities. (b) In a series of fourteen sera from cases (all males between the ages of 16 and 37 years) about which some knowledge of the dental state was obtained, a precipitin reaction was observed when serum-saline dilutions were shaken with an equal volume of dentinal "gen," and incubated at 37°C. for 24 hours. The "gen" was prepared by saline extraction of dentine suitably treated and diluted with 200 cc. of saline for each 1 gm. of dentine.

The extent of complement fixation with the same "gen" agreed relatively. There was little or no reaction with "gen" prepared from apparently normal dentine. Reactions with "gen" prepared from

[1] The original manuscript, of which this is the author's very brief abstract, was received for publication on March 21, 1933, and will be included in full in a later number in this volume.—[*Ed.*]

carious dentine agreed in intensity with the degree of apparent immunity to caries judged from the knowledge of the dental state and history, and have been classified as shown in Table 1. Forty-three sera (ex-Wassermann reaction), used to test these findings, showed that complement fixation in the presence of carious "gens" occurs independently of the Wassermann reaction. Precipitates obtained from carious "gens" fix 1 M.H.D. complement, but not 1.25 M.H.D. complement, in addition to fixation by the reagents used in their production.

3. It is suggested that the precipitate may account for the calcium barrier in the translucent layer of Tomes. It is conceivable that toxic

TABLE 1

Data on reactions with "gen" prepared from carious dentine

	NON-IMMUNE (SUSCEPTIBLE)	PARTIALLY IMMUNE	TOTALLY IMMUNE
Percentage of serum giving end-point of precipitin reaction against an equal volume of carious "gen"	3.5	1.3	0.15
M.H.D. complement fixed by 20 percent serum, in presence of one unit of carious "gen"	0–3	1.5–4.5	3–9

matter from the carious lesion enters the body through the pulp before the dentinal calcium barrier is laid down, and may even be the exciting cause of its deposition; and that repeated doses of toxic matter from successive carious lesions gradually stimulate the reactive forces of the body. The phenomenon of arrested caries may thus be capable of explanation; also the diminished liability to caries usually noticed with advancing years. It may be remembered also that calcareous deposits in response to stimuli from the site of a pathological process are by no means rare.

References to literature. (1) von Beust, Theo.: *Dental Cosmos*, 1912, *54*, 659. (2) Fish, E. W.: Medical Research Council Progress Report, 1926, App. iii.; May. (3) Fish, E. W.: An experimental investigation of enamel, dentine and the dental pulp, 1932; John Bale, Sons and Danielsson, Ltd., London. (4) Solis-Cohen, M.: *British Journal of Experimental Pathology*, 1926, *8*, 149.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the Louisville Section[1]
November 2, 1932

THEODORE B. BEUST, M.D., D.D.S., F.A.C.D., *Editor*

The second meeting,[2] of the Louisville Section was held at the School of Dentistry of the University of Louisville, November 2, 1932. *Chairman:* John T. O'Rourke, D.D.S., F.A.C.D., President of the Section.

Scientific program

I. Observations of the behavior of molten metal during casting. *Raymond E. Myers*, D.D.S., B.S. 275
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III. Prophylactic treatment of tooth tissue by means of silver nitrate. *Hugh Kellogg*, D.D.S., F.A.C.D. 279
IV. Condylar guidance versus incisal guidance. *W. M. Randall*, D.D.S., F.A.C.D. ... 279
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VI. Permanent records of the relations of teeth to selected points on the face. *Joseph E. Johnson*, D.D.S., F.A.C.D. 280
VII. Studies on calcium metabolism. *Alfred W. Homberger*, A.M., Ph.D. 280

I. OBSERVATIONS OF THE BEHAVIOR OF MOLTEN METAL DURING CASTING[3]

Raymond E. Myers, D.D.S., B.S., School of Dentistry, University of Louisville, Louisville, Ky.

The entrance of molten metal into the mold, its distribution throughout the pattern chamber, and the congealing of the metal, were seen in a laboratory experiment. This demonstration was accomplished by substituting a piece of transparent quartz[4] for one wall of an in-

[1] Received for publication on November 18, 1932.

[2] The first meeting (organization) was held on April 19, 1932.

[3] This paper is not identical with the one having a similar title in the proceedings of the eleventh general meeting of the International Association for Dental Research: *Journal of Dental Research*, 1933, xiii, p. 202; June.

[4] Credit is given to Mr. Harold M. Vernon for suggesting the use of quartz.

vestment mold. The coefficient of expansion with temperature, for quartz, is so low that it may be heated to redness and plunged into water without cracking; and therefore withstands the temperature of molten metal cast against its surface. Hence it was possible to observe, through a transparent quartz-plate, the behavior of the metal

FIG. 1

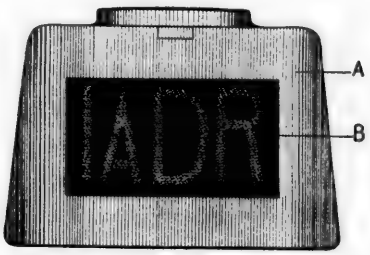

FIG. 3

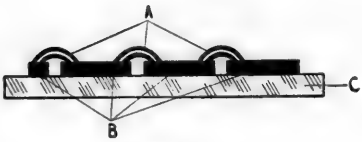

FIG. 2

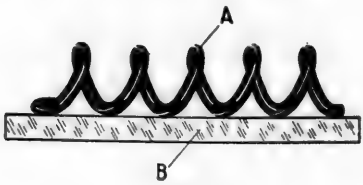

FIG. 4

FIG. 1. WAX LETTERS AND SPRUE-FORMER MOUNTED ON TRANSPARENT QUARTZ
A, Sprue-former. *B*, Quartz plate

FIG. 2. END-VIEW OF WAX LETTERS WITH CONNECTING LOOPS
A, Wax loops. *B*, Bases of wax letters. *C*, Quartz plate

FIG. 3. INVESTMENT FLASK WITH QUARTZ WINDOW EXPOSING PATTERN CHAMBER
A, Metal flask. *B*, Quartz window

FIG. 4. END-VIEW OF QUARTZ PLATE WITH WAX SPIRAL ATTACHED
A, Wax spiral. *B*, Quartz plate

during casting and congealing.[5] The wax pattern in this demonstration consisted of a group of four letters, I A D R (abbreviating

[5] An actual demonstration of this quartz-window casting was made by the author in July, 1930, before a group of the faculty at the School of Dentistry, University of Louisville; in December, 1930, before the Blue Grass Dental Society at Lexington; and in November, 1931, before the Odontological Society of Western Pennsylvania at Pittsburgh.

International Association for Dental Research). These letters, carved from a 12-gauge sheet of baseplate wax, were sealed to the surface of a quartz plate measuring $1\frac{1}{2}$ x $2\frac{1}{2}$ x $\frac{1}{8}$ inches. The pattern, with a wax sprue-former attached to the top of the I, is illustrated in *fig. 1.* To effect a time-interval between the filling of each letter with molten metal during casting, these letters were joined at their bases with looped pieces of wax concealed in the investment material. The connecting design is shown in *fig. 2.* The quartz plate bearing the wax was invested in a casting flask so that the face of the quartz would be adjacent to a square opening in the side of the metal flask. After the investment material had crystallized, that portion between the opening in the flask and the quartz plate was cut away, forming a window and permitting a clear view of the pattern chamber. The flask, showing the window, is illustrated in *fig. 3.* In a darkened room the metal was fused in the crucible and air pressure applied to effect the casting. The distribution of molten metal throughout the pattern chamber and its subsequent congealing were observed. This demonstration represents an experimental method of studying the behavior of molten metal under normal casting conditions.

Attention was called to the fact that the flow of metal in such a casting design is too rapid to permit adequate examination of the behavior of the metal.[6] This difficulty has been overcome, to a large extent, by resorting to novel casting designs. For example, velocity of the molten metal can be accurately measured by attaching a spiral wax pattern to the quartz plate so that the latter forms a tangent plane to the turns of the spiral (*fig. 4*), the flow of metal throughout the pattern chamber then requiring appreciable time and being visible only where turns of the spiral touch the quartz plate. Detailed reports of this work will be published in the near future. The following outline indicates some objectives: (1) *Rapidity with which molten metal enters mold and spreads throughout pattern chamber:* depends upon (a) number, (b) length, and (c) diameter of sprues; (d) temperature of flask, (e) and of molten metal; (f) porosity of investment material; and (g) character of pressure applied. (2) *Sequence of distribution of molten metal in pattern chamber:* depends upon (a) thickness of

[6] It is hoped that slow-motion pictures may be employed in these studies.

various areas of pattern chamber; and (b) diameter, and (c) placement and arrangement, of sprues. (3) *Comparative rates of congealing of different parts of casting:* depends upon (a) volume of metal in various areas of casting design, and (b) relation of sprues to casting design.

Addendum

On page 1010 of the *Journal of the American Dental Association* for June, 1933, there appeared an article by Dr. Wilmer Souder of the Bureau of Standards, entitled "Time required to cast dental restorations from molten alloy," in which he described the method of casting metal into molds provided with fused-quartz windows. The impression is left that the idea was original with Dr. Souder inasmuch as he gave no credit to anyone for having employed quartz windows to study behavior of molten metal during casting. As early as July, 1930, I demonstrated the method of substituting a fused-quartz plate for one wall of an investment mold before a group of the faculty at the School of Dentistry, University of Louisville. I also gave demonstrations of quartz-window casting before the Blue Grass Dental Society at Lexington, Ky., in December, 1930, and before the Odontological Society of Western Pennsylvania at Pittsburgh, in November, 1931. At the last-mentioned dental meeting, Dr. Souder also appeared on the program. I am not certain that the principle had not been used before I employed it in July, 1930, but I do know that it was then immediately adopted by a great number of men, and that it became widely known as an experimental procedure long before Dr. Souder read his paper at the meeting of the American Dental Association at Buffalo in September, 1932.

II. AN INVESTIGATION OF FORDYCE DISEASE

J. Irwin Essig, D.D.S., Louisville, Ky.

The author is studying cases of this unusual disease. Histological and pathological reports will be published as soon as available data permit.

III. PROPHYLACTIC TREATMENT OF TOOTH TISSUE BY MEANS OF SILVER NITRATE

Hugh Kellogg, D.D.S., F.A.C.D., Louisville, Ky.

The presentation was based on a method[7] in which the agent was sealed in artificially prepared cavities drilled between surfaces to be treated and the pulp. The extent of penetration was demonstrated in sections and on drawings. It was seen that tubules and inter-tubular substance, tufts and spindles, and interprismatic substance, were penetrated. Experiments showed that normal dentin, which is said to resist penetration, is actually permeable.

IV. CONDYLAR GUIDANCE VERSUS INCISAL GUIDANCE

W. M. Randall, D.D.S., F.A.C.D., University of Louisville, Louisville, Ky.

The author concludes, from clinical and experimental research, that proper determination of incisal guidance is more important to antagonization, with due regard to esthetics, than of the condylar guidance. It is recognized, however, that true and accurate relationship of these factors to each other and to cusp slant are essential. After the arrangement of anteriors has been made to conform to any desired overjet, overbite, or labio-lingual inclinations, and a careful grinding of the incisal edges has been completed, adjustment of incisal guidance on the articulator is undertaken to conform to conditions. Adaptations of registrations obtained from the patient's own mandibular movements are made after cusp slant, and condylar and incisal guidance, have been synchronized. Adjustments of inner and downward condylar guidance conform to the Gysi-gothic arch tracing. Esthetics correspond to views expressed by D. C. Campbell. The procedures call for very definite and characteristic incisal guidance instead of the arbitrary ajustments made heretofore, leading to more satisfactory esthetics, and to more accurate balance and function of the anterior teeth. Methods employing incisal guidance in the manner indicated have not heretofore been published.

[7] Clinics given before the American Dental Association at Washington, 1929; Denver, 1930; Memphis, 1931; Buffalo, 1932; also at the Chicago Mid-Winter Dental Meeting. 1929, 1930, and 1931.

V. INFLAMMATORY ZONES IN DENTIN[8]

Theodore B. Beust, M.D., D.D.S., F.A.C.D., University of Louisville, Louisville, Ky.

The disclosure by means of stains of inflammatory zones in dentin, previously described by the author, was discussed. The concept of "inflammation," as here applied, embraces any active biologic reaction of dentin to an irritant, such as caries. Photomicrographs illustrated the common occurrence of a distinct hyperchromophilic inflammatory zone completely isolated from the caries hearths. The observed phenomena appear to leave no doubt that these zones originate reflexly through reactions in the pulp.

VI. PERMANENT RECORDS OF THE RELATIONS OF TEETH TO SELECTED POINTS ON THE FACE

Joseph E. Johnson, D.D.S., F.A.C.D., Louisville, Ky.

The author described the use of a recording apparatus consisting of an instrument to ascertain and accurately correlate the four points selected for orientation of models; namely, inner canthus of the eye, and definite marks immediately in front of the tragi. With an instrument similar to a face bow, this record is transferred to a specially constructed articulator, which enables the operator to register any tooth movements occurring in three planes. Growth changes of the face may be recorded by this technic.

VII. STUDIES ON CALCIUM METABOLISM

Alfred W. Homberger, A.M., Ph.D., University of Louisville, Louisville, Ky.

The author discussed the relationship of calcium to normal bodily functions, and to skeletal structure. Experiments on rats, with diets containing various amounts of calcium in natural food products, to determine effects on tooth structure, are in progress.

[8] Paper read before the Research Section of the American Dental Association, Buffalo, N. Y., September, 1932. See also proceedings of the eleventh general meeting of the International Association for Dental Research: *Journal of Dental Research*, 1933, xiii, p. 186, June.

DENTAL EDUCATIONAL COUNCIL OF AMERICA

President's Address[1]

HENRY L. BANZHAF, B.S., D.D.S., F.A.C.D.
Marquette University, Milwaukee, Wis.

This is my first opportunity formally to express my gratitude to the members of the Council for the favorable reception given the recommendations in my annual address to this body at Buffalo last September.[2] At that time the activities and policies of the Council were being unjustly and maliciously assailed for political reasons, or for other motives more difficult to understand, by a small but exceedingly active group of individuals, each with his own selfish purpose. It became my duty, in the address to which I have alluded, officially to answer these attacks. Thanks to your active assistance and support, I am glad to record that much progress has been made in gaining for the Council the approval it deserves, and the recognition to which it is entitled. The House of Delegates of the American Dental Association on September 15, 1932, six days after the delivery of my Buffalo address, voted to ask the Trustees to reconsider the destructive resolution adopted in 1931, which was aimed to strip the Council of its duties and powers. On February 6, 1933, the Trustees concurred in this action and likewise rescinded the resolution, so that the Council has been reëstablished, as far as the American Dental Association is concerned, on the basis upon which it stood previous to the meeting at Memphis in 1931. Moreover, the National Association of Dental Examiners, at its annual meeting in September, 1931, adopted resolutions strongly endorsing the Council and pledging the Association's continued support. Even before the publication of my address, in the December number of the *Journal of Dental Research*, I received

[1] Read at the meeting of the Dental Educational Council of America, Stevens Hotel, Chicago, Ill., April 22, 1933.

[2] Banzhaf: *Journal of Dental Research*, 1932, xii, p. 887; Dec.

letters from many members of the dental profession, in all sections of the country, indicating in unmistakable terms their confidence in the Council, and their belief that dentistry should continue to be fostered and developed as an independent profession and in hearty coöperation with medicine. Many other things have happened of vital interest to dentistry during the period following our last meeting in Buffalo, but since you are all familiar with these events, and with the trends in our professional life, both alleged and actual, I need not enumerate them here.

At the last meeting of the American Dental Association efforts were made, by false leaders, to besmirch the fair name of the universities that are conducting dental schools, by ascribing to them impure and commercial motives. These ignoble efforts have failed miserably. The principal disparagement came from one whose courses for specialists have not been approved by either the Council or the Association of American Dental Schools because the motive of private profit in dental education, however valuable and worthy earlier in the life of the profession, is no longer commendable or respectable. The House of Delegates and the Board of Trustees of the American Dental Association were only temporarily misguided—a tribute to the high quality of their membership.

In several instances during the past year individuals with the best intentions and highest motives have unwittingly allowed themselves, although ignorant of the true conditions, to make careless statements, which are quite as damaging to the progress of the profession as those from malicious sources. A recent example of such a statement may be found on page 217 of the *Final Report of the Commission on Medical Education* (Dec. 1932), where the remarkable assertion is made that "it would seem logical that dentistry should be developed *under* medical education." I am unable to understand why such a statement was included in this voluminous report, for the Commission headed by Dr. A. Lawrence Lowell, President of Harvard University, could easily have learned otherwise by consultation with any representative group of dentists. Did the Commission study the dental problem sufficiently to justify it in expressing an opinion on this subject? Does it not seem reasonable that any question of importance in dental education may well be reserved for solution by dentists? As you

know, since 1930 the dental curriculum has been studied by a well-financed special commission composed of competent dental educators, expert educational advisers, and a trained executive secretary. This commission may be trusted to present data and conclusions for the guidance of all concerned. Dentistry has no occasion to suffer from an inferiority complex. What does the Commission on Medical Edution mean by the phrase "*under* medical education?" Is it not more than passing strange that two members of this Commission also signed the majority section of the *Final Report of the Committee on the Costs of Medical Care*, which comes to some important conclusions that are directly opposed to recommendations in the Medical Commission's report? Is it not also rather remarkable that, in the University of which Dr. Lowell is President, the same indifference prevails in the medical faculty regarding the need for knowledge of dental conditions by medical students as that existing in most medical schools. Concerning this interesting situation I quote from the Annual Report of the Carnegie Foundation for 1930:

"During the past year (1929–30) the Medical Faculty of Harvard University issued a volume of 194 pages entitled: *Synopsis of the Practice of Preventive Medicine as applied in the Basic Medical Sciences and Clinical Instruction of the Harvard Medical School.* Although the full professors in the Dental School at Harvard are members of the Medical Faculty, dentistry and all its medical aspects were overlooked in this important book. Medical schools . . . . are strangely unwilling even to teach the 'medical part' of dentistry to their students of medicine. . . . . If interest in the 'medical part' of dentistry continues at its present rate of growth (since 1924) in the medical schools in the United States, and if the number remains the same, then it may be expected that by 1985 all of the medical schools will devote an average total of eight (clock) hours in the entire curriculum to required work in dental and oral relationships."

I am sometimes comforted by the thought that these repeated attacks on dentistry are to be expected as a logical sequence of our *growing importance as a natural division of health service*, and mark a certain stage in the development of our profession. In the not distant future, when we shall have united on a standard dental curriculum, such curious propositions, because of the illogical premises upon which they are builded, and because of the absurdities to which

they would lead, will no longer be made. During the present state of uncertainty regarding the dental curriculum, even though these differences are more superficial than real, additional gratuitous advice may be expected from individuals who, although learned in their own vocations but knowing nothing or next to nothing about dentistry, are therefore not competent to tell us what to do.

May I point out briefly that in my opinion the imminent danger for dentistry now is no longer the direct dismemberment of dentistry into parts, with absorption of the "preferred" into medicine, and the "common" thrown on the market (which fantastic idea has been effectively exploded), but the actual *subordination* of dentistry to medicine, with all that such subjugation would imply in natural discontent for dentists and in serious demoralization of oral health service. Dentistry, by reason of its importance, is entitled to a place in the sun, and this subordination-idea must be overthrown before it gets effectively under way. It behooves us to watch our step in our efforts to establish coördination with medicine, so that the *coördination* which is so much to be desired cannot by any possible chance become *subordination*. Statements concerning dentistry, such as that published in the *Final Report of the Commission on Medical Education*, should be challenged, and shown to be illogical, just as soon as they are made.

The period of stress through which each member of the profession is passing, and the difficulties he encounters in his daily experiences as a dentist, as well as the problems that are being met by such dental organizations as the component groups of the American Dental Association, and by such organizations of national significance as our own, and educational institutions in general, will undoubtedly prove to be blessings in disguise. Man does not develop to his fullest capacity until he has experienced disappointments and reverses—until his ideals are in danger, or the possibility of complete destruction of institutions builded by his forefathers looms in sight. Particularly is this true of the young men in the profession—those who are most richly endowed with undepleted energy, and who up to now have been passive onlookers. The emergency we are passing through is arousing them, and prompting them to assume positions of leadership. Individual members of the profession as well as all dental groups, including this

Council, have been stimulated to prove the worth of dentistry as a co-partner of the professions engaged in health service.

I appreciate the fact that we have an exceptionally heavy program at this meeting, and that brevity in this address is desirable. Therefore I shall discuss the merits of only the first item in the call for this meeting. The Secretary informs us that requests for inspections have been received from the Deans or Acting Deans of four dental schools who believe they have met the standards for a higher classification. These schools are the Columbia University School of Dental and Oral Surgery, the Dental Department of Georgetown University, the College of Dentistry of the University of Nebraska, and the Dental School of Temple University. The application from Columbia University is especially appreciated by members of the Council, because it indicates a gratifying change of general policy since our last meeting.[3] In my address at Buffalo last September I made the following statement, which was approved by the Council at that time: "We should not, however, pending the report of the Committee of the American Association of Dental Schools on the Survey of the Dental Curriculum, make any new suggestions to dental schools, or regulations for them, or inspect them, except at their request individually. To do so would be illogical, since the standards we might set up, so far as they relate to the curriculum, may soon be changed or modified by the Committee's findings." From correspondence I have recently had with the Executive Secretary of this Committee, it appears that the final report will not be made in the near future, and that more time is needed to complete the work upon which an acceptable final report may be formulated. Under the circumstances, I am of the opinion that to neglect to inspect these schools in the near future would subject them to an injustice which, I am sure, no member of the Council intends. Under normal circumstances a reasonable delay might be endured by the schools, but the changes that have taken place recently in our whole social order have made it expedient for us to do things which have never been done before, and for which there is no established precedent. Because the Council is temporarily without funds to pay the expenses incident to the inspections, I believe that the

[3] Banzhaf: *Journal of Dental Research*, 1932, xii, p. 892; Dec.

rule forbidding schools to pay these expenses should be abrogated until the financial condition of the Council assumes normalcy. I also believe that, in the interest of economy, the inspecting committee for each school may well be limited to two members instead of the customary three. The whole Council may then be consulted by mail, and a decision promptly made upon the recommendation and written report of the Committee. The authorities of these four schools, believing they have made progress toward a higher classification, are looking to us for recognition of their accomplishments. If the progress claimed in each case is found to meet the reasonable standards we have set up, there now seems no good reason why action by the Council should be further delayed.

I wish to make a recommendation concerning a matter not mentioned in the call for this meeting. It is obvious to all who have a full understanding of the work this Council is endeavoring to do in behalf of dental education, and the responsibilities with which it is charged, that in order to function most effectively the services of an Executive Secretary must be employed. Our worthy Secretary, Dr. Albert L. Midgley, has carried the burdens of his office much longer than we have any right to expect, and it is only because of his loyal devotion to the cause of dental education that he has been able, for nearly fifteen years, to do his work with exceptional energy and enthusiasm. We do not wish to lose his services, and therefore he should be given the assistance of an efficient and competent Executive Secretary. Such an Executive Secretary, in order to be of greatest service to the Council, and through it to dental education, should necessarily be possessed of rare gifts for organization and investigation. He should have a thorough scientific background, and, in addition, should have lived in the work long enough to acquire a working knowledge of dental education and the problems confronting dental schools. I, therefore, suggest that we present, to the American Dental Association or other organizations, a persuasive appeal for financial assistance for the definite purpose of paying the salary and incidental expenses of an Executive Secretary.

I have already referred to the spirit of unselfish service that has always been manifested by the members of the Dental Educational Council of America when there is work to be done, as there was last

September. There is one individual, however, who is neither a member of the Council nor a member of the dental profession, whose activities during the past ten years in behalf of dental education and dental research probably exceeds that of any other person. I refer to our friend Dr. William J. Gies, Professor of Biochemistry in the School of Medicine of Columbia University. I am sure that although each member of the Council is fully cognizant of his great services in behalf of dentistry, they will take genuine pleasure in hearing them reviewed briefly in order to do him honor. While I cannot, in the time at my disposal, touch even the high spots in his career as it relates to dentistry, it has occurred to me that it would be possible to give a brief account of the work he has been engaged in during a limited time, such as the past eight months, as being representative of his activities during any other period of similar length. In the October (1932) number of the *Journal of Dental Research*, he published at his own expense his paper entitled: Comment on some misstatements regarding the Carnegie Foundation's study of dental education." He made preparations for the meeting of the American College of Dentists in association with the American Association for the Advancement of Science, at Atlantic City on December 30, and took an active part in the meeting. He published his paper on "The status of dentistry," in the December number of the *Journal of Dental Research*—a critical analysis, which is considered by many to be the most outstanding paper that has ever been published on this subject. In this paper he used the expression "independence with interdependence" in describing the ideal relationship of dentistry to all accredited professions of health service, including medicine. His conclusions in this remarkable argument are unanswerable, and should settle for all time in the affirmative the question as to whether dentistry should continue to be developed as a separately organized profession. Dr. Gies corresponded with members of the Committee on the Costs of Medical Care, and with many dentists in the United States and Canada, regarding the misrepresentations of dentistry in the *Final Report* of that Committee, and published a complete report of the whole matter in the February (1933) issue of the *Journal of Dental Research*. He corresponded with members of the Commission on Medical Education regarding a revolutionary recommendation about dentistry in the Commission's *Final*

Report, and published an analysis of the same in the February number of the *Journal of Dental Research*. He conducted the arrangements for the program and meeting of the International Association for Dental Research, on March 18 and 19. On page 1129 of the April 8, 1933, issue of the *Journal of the American Medical Association*, will be found a letter from Dr. Gies to Dr. Morris Fishbein, Editor, in which a reply is made to a very damaging review in the issue of April 23, 1932, of that publication, and which had endorsed some of the statements made in the pamphlet on "Some phases of dental education in the United States." Dr. Gies, if my information is correct, is at present engaged in obtaining and editing, for publication, a large number of abstracts for the Proceedings of the International Association for Dental Research.[4] In addition to all these labors, he edited each successive bi-monthly issue of the *Journal of Dental Research*. Dentistry indeed has a devoted friend in William J. Gies. He has stepped into the breach whenever the profession needed a champion. There are none here at this meeting today who do not feel for him sentiments of the deepest gratitude and affection.

In conclusion, I desire to express my personal appreciation for the loyalty and devotion each of you has shown to the Council. As you know, I have been a member of this body since the Council's organization twenty-four years ago, and have had the honor of serving as its Secretary and its President. During these many years I have been associated with many men who served on the Council for various periods, and can truthfully say that almost without exception these men were always ready to make personal sacrifices in order that the work might go on. May the same enthusiasm for progress that has been the distinctive characteristic of the Council since its organization in 1909 never be absent in future years.

[4] Proceedings of the International Association for Dental Research: *Journal of Dental Research*, 1933, xiii, p. 173.

AMERICAN ASSOCIATION OF DENTAL EDITORS[1]

Proceedings of the Second General Meeting
Buffalo, N. Y., September 10, 1932

ROBERT S. CATHERON, D.M.D., *Secretary*
Boston, Massachusetts

I. ABSTRACT OF THE MINUTES

The second general meeting of the American Association of Dental Editors was held at the Hotel Statler, Buffalo, New York, September 10, 1932. The following members attended one or more of the three sessions: G. M. Budlong, *Florida D. Jour.;* R. S. Catheron, *Bull. Mass. State D. Soc.;* F. B. Clemmer, *Ill. D. Jour.;* W. R. Davis, *Mich. State D. Soc. Bull.;* C. W. Freeman, *Bull. Chicago D. Soc.;* H. E. Friesell, *D. Educ. Council (J.D.R.);* W. J. Gies, *Jour. D. Res.;* J. E. Gurley, *Jour. Cal. State D. Assoc.;* G. F. Hale, *Bull. N. C. D. Soc.;* K. F. Knudtzon, *Bull. D. Alumni Assoc., Univ. Ill.;* A. L. Midgley, *Amer. Coll. D. (J.D.R.);* J. T. O'Rourke, *Ky. State D. Assoc. Bull.;* B. B. Palmer, *N. Y. Sec. Internat. Assoc. D. Res. (J.D.R.);* Grace R. Spalding, *Jour. Periodont.;* W. F. Swanson, *Bull. Odont. Soc. W. Penn.;* E. A. Thomas, *Jour. Neb. State D. Soc.;* L. E. Van Kirk, *Pittsburgh Sec. Internat. Assoc. D. Res. (J.D.R.);* R. S. Vinsant, *Amer. Assoc. D. Schools;* W. A. Wilson, *N. J. State D. Jour.* In addition three applicants for membership were present: O. W. Brandhorst, *Bull. Mo. State D. Soc.;* U. G. Rickert, *Jour. Mich. State D. Soc.;* C. L. Smith, *Bull. Dental Soc. D.C.*

The *morning* was devoted to committee meetings and to an informal conference at which President Thomas showed a number of ingenious and economical aids in filing and recording. The *afternoon* session was begun with President Thomas in the chair. In the absence of Dr. M. W. Carr, Secretary, Dr. R. S. Catheron served as Secretary

[1] The proceedings of the first general meeting (January 18, 1932) were published in the *Journal of Dental Research*, 1932, xii, p. 223; April.

pro tem. The President's opening address, on dental journalism, and a paper by Dr. Catheron, on the problems of a dental editor, were followed by reports of committees and of the Treasurer. The following applicants for active and associate membership were elected: O. W. Brandhorst, *Mo. State D. Assoc. Bull.;* G. C. Douglass, *Bull. Second (N. Y.) Dist. D. Soc.;* H. S. Glick, *Bull. Alumni Assoc. Coll. D., Univ. Cal.;* C. E. Hatch, *Tufts. D. Outlook;* M. K. Hine, *Bull. Alumni Assoc. Univ. Ill.;* M. H. Jeffrys, *D. Hygiene Quar.;* H. J. Leonard, *Columbia D. Rev.;* W. C. McBride, *Mich. State D. Bull.;* F. J. McLaren, Jr., *Bull. Second (N. Y.) Dist. D. Soc.;* A. H. Merritt, *Jour. Periodont.;* E. E. Parkinson, *Wis. D. Rev.;* F. G. Rea, *Bull. Second (N. Y.) Dist. D. Soc.;* Lewis Rice, *D. Outlook;* U. G. Rickert, *Mich. State D. Soc. Jour.;* Stella Risser, *D. Bull. Houston D. Soc.;* T. P. Ryan, *Jour. Minn. State D. Assoc.;* H. M. Seldin, *Bull. Second Dist. (N. Y.) D. Soc.;* Jacob Shapiro, *Bull. Second Dist. (N. Y.) D. Soc.;* C. L. Smith, *Bull. Dent. S. D. C.;* W. A. Wilson, *Bull. Hudson Co. (N. J.) D. Soc.* After discussions and actions on a variety of official matters, the following officers were elected for 1932–33: *President*—E. A. Thomas. *Vice-president*—W. R. Davis. *Secretary*—R. S. Catheron. *Treasurer*—C. W. Freeman. *Editor*—Grace R. Spalding.

In the *evening*, an informal dinner was held at the Hotel Statler, President Thomas in the chair. Dr. Morris Fishbein, who had accepted the invitation to be our guest speaker, and to address us on "The leadership responsibility of the editors of professional journals," was subsequently invited to speak before the American Dental Association later in the week. As it was obviously desirable that the greater number should have the opportunity to hear him, the Association surrendered its prior claim. Dr. William J. Gies, who was to have discussed Dr. Fishbein's paper, kindly consented at short notice to take Dr. Fishbein's place. Dr. Samuel M. Gordon discussed the work of the Council on Dental Therapeutics.

II. QUOTATIONS FROM THE PRESIDENT'S ADDRESS AND FROM REPORTS

1. President's address

. . . . The speaker believes we have reached an age when we are able to walk alone and fulfill our duty to support a professionally owned journalism. . . . .

The non-proprietary dental periodical issued by a dental society has certain definite obligations to that society. It should report the society's activities, and keep its readers in touch with the work of the organization and of fellow members. It should advise them of the work done and being done by similar organizations, within or without the profession, throughout the world. It must be representative of the scientific, economic, educational, legislative, and welfare phases of dentistry. It must be coöperative, not individual. It must preserve the dental history of its society for the benefit of future historians. It must maintain the highest scientific and professional standards. The advertising should be truthful and useful. It must lead its society, not follow; inspire, not only reflect. It must preach progress and educate to the full extent of its influence. It must be interesting, attractive in appearance, and well edited. The degree to which this is accomplished depends on the editor and his associates. A balance must be maintained between income and expenditures. Fortunate is the editor whose hands are not tied; fortunate is the journal which does not expect income from sources that do not express the highest ideals of a majority of the members of its society, not only in all forms of professional ethics, but in advertising ethics—a journal whose ownership is divorced from all outside business interests, and which is managed and controlled by dental practitioners. Such a journal is one that deserves support from our dental authorities, writers, and contributors. Such a journal can fearlessly discuss and expose the schemes which are, and always will be from time to time, devised for the furtherance of individual interests at the expense of the good name of dentistry. A society dental journal is the official publication of its society. It is the organ of that society and should reflect the highest ideals of the dental profession within the territory it serves. Dentists actually read journals of this kind. If they did not, we would have stopped publishing them long ago, and new ones would not be appearing each year. There is an actual need for them; and as long as they go on filling that need, they will continue to be published. They will also continue to be improved as long as the editors have the wisdom to see and then to meet the needs as they arise. The chief duty of the editor is to edit. He must see that each article is concrete; that it expresses exactly in a clear, concise

style, what the author evidently wishes to say. It is the editor's duty to save time for the reader and not permit the contributor to waste it. The editor's primary duty is to the reader, not to the contributor. . . . .

The *proprietary* journal has a monetary consideration back of it. Its editor and manager are inspired to effort as participants in the monetary revenue resulting therefrom. The editor and manager of a *non-proprietary* journal must give time and effort from their busy practices, and receive in return only the satisfaction of tasks well done. . . . . Keen competition is causing much rivalry in all lines of business, and this is leading to frequent distortion of truth in the claims and statements made to advertise various proprietary articles and commodities. The confusion of the public mind relative to the differentiation between a profession and a business is derogatory to a healthy professional order, and must be considered very carefully at this time. . . . . Only a small percentage of dentists attend our conventions, and dentistry has grown to such proportions that to present a balanced program, a variety of subjects must be covered in their many phases. As a consequence, those who attend go home with a hazy outline of what they have seen and heard. The society journal has the opportunity to present the convention benefits for study, not only for those who attend, but also for those who stayed at home. The journal stimulates a greater interest in organized dentistry, develops interest in other members and their activities, increases interest in the dentist as such, and relates his services to humanity. . . . .

If any one group of men in the dental profession needs to be organized, helped and encouraged, it is the dental editors. If it is true that our newspapers and similar periodicals mold, shape, and influence our public life, particularly the small-community papers, it is apparent that even the small dental journal has no mean place in molding and directing dental progress. . . . We are here to serve the profession of dentistry—not only as well as those of the past have served, but better—by organized action in helping (a) the individual editor and thereby his journal, (b) the dentist by his perusal of the editor's efforts, (c) the profession at large, and (d) the public it serves. The banding together of this small group, representing the dental com-

munity-interests of America, will not only afford personal benefits, but will inspire greater interest among dentists in the local journals and in their standing compared with others. This will lend moral support, and also will stimulate financial appropriations, for the furtherance of professional journalism. . . . .

2. Committee on dental literature

. . . . Your Committee appreciates the urgent need for constructive criticism of dental literature, which, although steadily being improved, has not yet attained an excellence that is commensurate with the importance, responsibility, and opportunity of the dental profession. We believe our first study should aim to bring about marked improvement of the *non-proprietary* dental journals in the United States. We suggest that your Committee could be most useful in this relation, during the coming year, in studies of the journals (*represented in our Association*) whose editors would express a *desire* to receive the Committee's frank constructive comment on the literary quality of their periodicals. Such a "literary clinic," applied to volunteers and within our own family so to speak, would afford a very favorable basis on which to initiate the Committee's work. It would also, from the mutual friendliness of all concerned, open the doors widely for the development of a new technique for literary inquiry. It would give your Committee the practical experience and understanding that would probably enable the Committee to conduct most effectively all phases of its future effort. . . . . *Grace R. Spalding, Walter Hyde, W. J. Gies, Committee.*

3. Committee on advertising

. . . . *Conclusions.* A. Acceptance of advertisements by a non-proprietary dental periodical does not necessarily alter in any way its non-proprietary status, which is based on its ownership. (1) It is expedient, proper, and equitable that dental trade-houses, and others having merchandise or services to sell to the dental profession, be afforded ample opportunity to present their claims for patronage through the advertising pages of the accredited dental periodicals. (2) If it be true, as seems to be generally agreed, that dental periodicals offer the best means to introduce merchandise to the profession,

and that the trend is toward elimination of proprietary dental journalism, then it would be neither logical nor fair to close the pages of the non-proprietary periodicals to proper advertisements.

B. The non-proprietary periodicals are in a position of great responsibility in respect to the standards they establish for the censorship of advertisements. (1) A profession is judged to a marked degree by its literature. If responsible dental periodicals contain advertisements of false or fraudulent claims for preparations of secret formulae, or of unknown or unproven therapeutic value, or if they contain advertisements of products known to be actually harmful, or if they contain advertisements presented in an exaggerated, undignified or otherwise inappropriate form, then the periodicals and the profession they represent suffer serious loss of prestige, with all the accompanying liabilities.

Recommendations. A. Your Committee recommends that the Association adopt the following code as representing its views on the acceptability of advertisements; and that the officers of the Association be instructed to endeavor to secure approval of the code by the owners of all non-proprietary dental periodicals: (1) Only such therapeutic remedies or dentifrices as have been approved by the Council on Dental Therapeutics of the American Dental Association shall be advertised. (2) Advertisements of products not under the jurisdiction of the said Council shall be acceptable only if they meet the requirements of (a) truth in statement, (b) reasonableness in claim, and (c) dignity in presentation. (3) Advertisements shall be confined to the advertising section, to the inside and outside of the back cover, and to the inside of the front cover. In keeping with this provision, advertisements shall not be distributed on or among the pages devoted to professional affairs. (4) Complimentary references to individual advertisers or their products (commonly referred to as "puffs" or "boosts") shall be avoided, but it shall not be considered improper to refer to the advertisers as a group. (5) Only such advertisements shall be acceptable as are frankly presented *as advertisements*. No advertisements shall be acceptable that have the appearance of being scientific articles, editorials, or anything else than advertisements, for they would not meet the requirements of honesty and integrity specified in A, (2), above.

B. It is recommended that the foregoing code be adopted to become effective as of January 1, 1934, or sooner if practicable; and that no new contracts for advertisements that would be ineligible under the code be made thereafter. *F. B. Clemmer, C. S. Foster, B. B. Palmer, Committee.*

4. Committee on coöperation

. . . . *Suggestions.* (1) Establishment of a central office for dissemination of news, cuts, and information for editors, or enlargement of the Secretary's office. (2) United action by editors of the Association to give publicity to deserving projects, such as the dental examination for life insurance and unethical advertising. (3) Coöperation for use of cuts and of editorials. (4) A page or two containing condensed summaries of articles appearing in the leading medical and dental journals both in the United States and foreign publications. (5) Read several good books on journalism—or better still, take one or more courses in journalism either directly or by correspondence . . . . *Claude Wood, G. M. Budlong, K. F. Knudtzon, Committee.*

JOURNAL OF DENTAL RESEARCH

WILLIAM J. GIES, F.A.C.D.

Editorial office, Journal of Dental Research, New York City

I. PROSPECTIVE CHANGE OF OWNERSHIP

The *Journal of Dental Research* was not founded for financial profit. The *Journal* has never been a commercial project. In 1919, when the *Journal* was first issued, no dental society cared to conduct it. At the beginning we informally expressed the hope that successful performance of the *Journal's* desirable functions would soon induce at least one important dental organization to desire to own the *Journal* and to assure its permanence. Since 1919 we have repeatedly stated that the name and good will of the *Journal* is transferable, without any pecuniary or personal reservation, to a leading dental society. Since 1929, annual reports to the American College of Dentists, by its Commission on Journalism, have stimulated the interest of several dental associations in the future ownership of the *Journal*. The Commission's public report in 1932[1] facilitated presentation of proposals relating to transfer of ownership by the (a) American Dental Association (May 22), the (b) International Association for Dental Research with the coöperation of the American College of Dentists and the New York Academy of Dentistry (May 26), and the (c) First District Dental Society of the State of New York (June 16). These three proposals are now being studied in detail by all the editors.

II. CORRECTION OF MISSTATEMENTS ABOUT THE JOURNAL

We have endeavored to make the *Journal of Dental Research* a cumulatively useful agency for the advancement of dental research and the support of idealism in dentistry; and for the ensuing furtherance of oral health-service, the enhancement of the usefulness, appreciation, and dignity of the dental profession, and the promotion of the

[1] The status of dental journalism in the United States. Report of the Commission on Journalism of the American College of Dentists (1928–1931), B. B. Palmer, Chairman; pp. 238. Reference to the *Journal of Dental Research*: pp. 171–176.

public welfare. The many informal discussions among dentists regarding ultimate transfer of ownership of the *Journal* to a leading dental organization or group of such societies, in recent years especially, have been conducted with gratifying recognition of these efforts. A few misapprehensions that have come to our attention were promptly corrected by informed persons. Several representations were so obviously inaccurate or unjustified that they have been generally disregarded. We are obliged, however, to correct plausible misstatements, by two speakers, as recorded on pages 137–139 of the *Transactions of the Seventy-fourth Annual Session of the American Dental Association* (Buffalo, September, 1932).

The comment by one of the speakers, as reported in the *Transactions*, included these allegations (page 137): ". . . . We have information that the Journal of [Dental] Research at the Columbus, Ohio [1932] meeting [of the International Association for Dental Research] reported that they were contemplating abandoning this Journal, or that the editor has seen fit to discontinue or drop the issue and they are looking about for a reputable organization or some dental journal to take over this Journal." All of which is a conglomeration of untruths. The speaker, instead of stating the truth after making suitable inquiry where the facts were readily obtainable, was content to comment irresponsibly. The context shows that this speaker was unaware of his ignorance, and had no malicious purpose. We did not communicate with him about this situation because we are confident his intentions were honorable, and we believed the prospective corrections of the second speaker's misrepresentations would cover those of the first. Shortly after we read the published comment by the second speaker, we sent him (April 19) a summary of inexcusable assertions in his recorded remarks, which included the following specific misstatements of fact (pp. 138–139):

". . . . The Journal of Dental Research is owned by the First District Dental Society. . . . .If you look at the first page [cover?], you will find that the First District Dental Society is the owner. The legal rights are vested in the First District Dental Society. . . . . The First District Dental Society controls a journal known as the Journal of [the] Allied [Dental] Societies [continued in the Journal of Dental Research]. Rights of ownership, according to the Constitution and By-laws of the First District Dental

Society are vested in the First District Dental Society. Any society which thinks it can take it [Journal of Dental Research] over without consulting the First District Dental Society is going to be up against a legal complication. . . . . The Journal [of Dental Research] could not exist at all without that [financial support of the First District Dental Society]. . . . . In the letter we received from Dr. Gies, he said the Journal was very much a white elephant for a number of years. . . . . If the American Dental Association wants the Journal of Dental Research, the First District Dental Society can get control and give it to you, but it has to come through the First District Dental Society because they have vested property rights and ownership in that Journal. . . . . It [Journal of Dental Research] evidently is free for the asking and anybody can take it."

The directly contradictory character of some of these untruthful remarks is self-evident. This fact suggests that the speaker was incorrectly quoted in the published record. Printer's proof of the comment for the official transactions is regularly submitted by the Secretary of the American Dental Association to the respective speakers, who thus receive ample opportunity to confirm or correct the stenographic reports. There is no doubt of the correctness of the record containing the above quotations. In our comment on this array of untruths and others in the record related to them, we wrote, in a private letter to their author, in part as follows (April 28):

"The *Transactions of the American Dental Association* is presumably a reliable periodical publication. The American Dental Association is avowedly a responsible body. . . . . All uninformed persons who read, in the *Transactions*, the comment ascribed to you . . . . will, or may, believe it. *All the quotations from the statement publicly ascribed to you that are summarized above are untrue. Other remarks in the comment publicly ascribed to you are misleading.* The statement as a whole represents you as seeking, with the aid of false allegations, to prevent proposed action affecting ownership and management of the *Journal*. These untruthful assertions were, and are, seriously injurious to the *Journal*. . . . . The libelous statement [as a whole] is now a readily accessible public record. Regardless of the reasons for the untruths in that statement, *prompt and effective reparation is required.* I now formally ask, on behalf of the *Journal*, that that reparation be given the form of a voluntary public correction of all the misrepresentations in the statement, including also a clear indication that none of the unfair implications was intended" . . . .

To this we received the following reply (May 5):

"In reply to your letter of April 28, I am preparing a correction of the report of the transactions published in the *Journal of the American Dental Association* which I will forward to . . . . , requesting that same be published in the June issue, if possible. Do you desire a correction to be published in the *Journal of Dental Research,* as coming from me? If you so desire, I will be glad to send it to you."

To this very satisfactory expression of purpose to make the reparation requested, we sent the following response (May 6):

"Your favor of the 5th of May was received this afternoon. The *Journal of Dental Research* should publish your correction. *To save you unnecessary trouble,* I suggest that we publish the statement you expect to send to . . . . , provided this would be agreeable to him. If . . . . should print it before August—I hope it will be possible to include it in their June number, as you suggest—the version of it in our August number would be given as "reprinted from the . . . ." This procedure would make one correction suffice for all purposes. If, however, you prefer to prepare a special statement for the *Journal of Dental Research,* this alternative would be entirely satisfactory."

In the concluding reply from our correspondent, indicating full agreement between us on a reparative procedure intended to serve his convenience, he wrote (May 9):

"In reply to your letter of May 6, I am quite sure the correction I sent to . . . . will be published before August. If it does not get in the June issue, it will get in the July issue. I am sure that . . . . will be perfectly willing for you to publish it in the *Journal of Dental Research.*"[2]

The present note impersonally calls direct attention to some details in gross misstatements about the *Journal of Dental Research,* in the *Transactions of the American Dental Association,* and protects the *Journal of Dental Research* against the consequences of any future misjudgments that the said published misrepresentations might occasion or support.

[2] The issue of the *Journal of the American Dental Association* for June, 1933, contains, on page 1090, an editorial reference to this situation under the title: "A correction of statements regarding the *Journal of Dental Research.*" Our correspondent's communication was not included and, for that reason, is not reprinted here.

CALCIFICATION OF TEETH AND BONES ON RACHITIC AND NON-RACHITIC DIETS[1]

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When the present investigation was started, it seemed probable that alterations in Ca, P, and ash of incisors would occur in rats on a rachitic diet known to produce marked changes in bone. Assuming that such changes would be produced, it was planned to determine whether teeth would respond to preventive and curative agents effective for bone.

Experimental. The rachitic diet employed was Ration 2965 of Steenbock and Black (1), as modified by Epstein (2), and consisted of yellow cornmeal, 66 percent; wheat gluten, 20 percent; egg albumin, 10 percent; $CaCO_3$, 3 percent; NaCl, 1 percent; spinach, 10 grams per rat per day; and water, *ad lib.* Excluding the spinach, this diet contains 1.236 grams of Ca and 0.17 grams of P, in 100 grams—a Ca:P ratio of 7.27. Young albino rats, 35–44 days old, were used; they developed marked rickets on this diet, accompanied by consistent growth. Addition of 2 percent KH_2PO_4, or 2 percent cod-liver oil, makes the diet non-rachitic. The 2 percent KH_2PO_4 supplement reduces the Ca:P ratio from 7.27 to 1.93. Ca, P, and ash of the incisors and tibias were compared on five diets, in two groups: *Group I.*—(1) Rachitic; (2) rachitic, plus KH_2PO_4 (2 percent); (3) rachitic, plus KH_2PO_4 (2 percent), plus cod-liver oil (2 percent). *Group II.*—(4) Rachitic, plus KH_2PO_4 (2 percent); (5) rachitic, supplemented after 30–45 days by KH_2PO_4 (2 percent); (6) rachitic, supplemented after 30–45 days by cod-liver oil (2 percent); (7) rachitic, plus KH_2PO_4 (2 percent), plus cod-liver oil (2 percent). Diets numbered (4) and (7) in Group II are the same, respectively, as (2) and (3) in Group I, and were included so that the rachitic

[1] This work was conducted with the aid of a grant from the Chemical Foundation for research in biochemistry. A preliminary report was presented at a meeting of the New York Section of the International Association for Dental Research, March 13, 1930: *Journal of Dental Research*, 1930, x, p. 267.

animals receiving supplements of KH_2PO_4 and cod-liver oil might be compared with non-rachitic rats of similar ages. Litter-mates were used in each comparative group. There were at least six rats on each diet; twelve, on Diet 1; twelve on Diet 2.

After 30–45 days the rats in Group I were killed with chloroform, and the incisors and tibias obtained. The fresh materials were then weighed, pulps removed with a dental pulp extractor, and bones split and marrow removed. The rats in Group II were killed after 64–80 days, and teeth and bones similarly treated. Most of the specimens were analyzed for Ca and PO_4 by the method of Shear and Kramer (3). The tissues were crushed, and extracted for four hours in a Soxhlet extractor with a mixture of equal parts of alcohol and ether. The air-dried residue was then pulverized to pass an 80-mesh sieve, and dried at 100°C. for 24 hours. Approximately 50-mg. portions were digested for ten minutes with 5 cc. of 1*N* HCl;

TABLE 1

Average daily gain in weight, and average weight of incisors and tibias, of rats in Group I

DIET	BODY	TEETH	BONES
	Daily gain in 30–45 days	Weight of 4 incisors	Weight of tibias
	grams	*grams*	*grams*
(1) Rachitic	1.03	0.173	0.222
(2) Rachitic + KH_2PO_4	1.50	0.192	0.273
(3) Rachitic + KH_2PO_4 + cod-liver oil	2.14	0.254	0.398

after filtration, Ca and PO_4 were determined in the HCl solution. The remainders of the pulverized and extracted materials were ashed (in platinum crucibles), and Ca and PO_4 determined.

Results. Considering the severity of the rachitic diet, the animals grew well. Addition of KH_2PO_4 (2 percent) resulted in increased growth, which was further improved by addition of cod-liver oil (2 percent). These data, and the average weights of fresh teeth and bones, are given in *table 1*, where it may be noted that the average weights of teeth and bones roughly parallel the gains in body weight. Despite the fact that marked reductions in Ca, P, and ash were produced in the bones on the rachitic diet, no significant reductions were produced in the teeth. The average values, and average deviations of Ca and P of *unashed* teeth and bones of rats on diets in Group I, are

given in *table 2*. The values for the tissues analyzed *after being ashed* are given in *table 3*. The results obtained by the two methods are in

TABLE 2

Averages and average deviations for Ca and P on diets in Group I. (Figures represent percentage of dry weight of alcohol-ether extracted material)

DIET	TEETH		BONES	
	Ca	P	Ca	P
(1) Rachitic	26.7±0.6	13.8±0.5	11.3±1.2	5.9±0.8
(2) Rachitic + KH_2PO_4	26.2±0.8	13.8±0.6	20.3±0.7	9.7±0.4
(3) Rachitic + KH_2PO_4 + cod-liver oil	26.5±0.5	14.0±0.1	21.8±0.7	10.5±0.5

TABLES 3 AND 4

Averages and average deviations for ash, Ca, and P on diets in Groups I and II. (Figures represent percentage of dry weight of alcohol-ether extracted material)

DIET	TEETH			BONES		
	Ash	Ca	P	Ash	Ca	P
Table 3. Group I						
(1) Rachitic	73.8±1.1	26.5±0.7	14.2±0.4	40.0±2.4	13.7±2.5	6.8±0.4
(2) Rachitic + KH_2PO_4	73.2±0.8	26.2±0.8	14.3±0.7	56.3±1.8	21.0±1.8	10.0±0.3
(3) Rachitic + KH_2PO_4 + cod-liver oil	73.0±0.9	26.3±0.4	14.1±0.6	58.1±2.8	21.6±0.8	10.7±0.5
Table 4. Group II						
(4) Rachitic + KH_2PO_4		26.3±0.5	13.6±0.3	...	22.2±0.6	10.4±0.4
(5) Rachitic, supplemented after 30–45 days by KH_2PO_4		26.1±0.3	13.6±0.3	...	20.1±1.3	9.4±0.6
(6) Rachitic, supplemented after 30–45 days by cod-liver oil		26.7±0.6	13.9±0.6	...	20.4±0.8	9.4±0.5
(7) Rachitic + KH_2PO_4 + cod-liver oil		27.2±0.6	14.4±0.5	...	23.8±1.2	11.2±0.5

Diets (2) and (3) are the same as (4) and (7), respectively

close agreement. On Diet 1, the bone P was reduced 49 percent, the bone Ca 48 percent, as compared with the non-rachitic Diet 3; while the teeth were not significantly different on the three diets. As

would be expected, the bones gave greatly reduced values on the rachitic diet, but were improved by addition of 2 percent of KH_2PO_4—more markedly by further addition of 2 percent of cod-liver oil.

Since the rachitic diet failed to alter the composition of teeth with respect to ash, Ca and P, the second part of this study, involving recovery (diets in Group II), added nothing with regard to teeth. It is interesting to note, however, that although addition of cod-liver oil to the rachitic-diet-plus-KH_2PO_4 (in Group I) produced no change in teeth in 30–45 days, it caused a slight increase (in Group II) in 64–80 days. When 2 percent KH_2PO_4, or 2 percent cod-liver oil, was added after the rats had been on the rachitic diet for 30–45 days (Diets 5 and 6), the Ca and P were increased in the bones to about the same level in each case, but fell somewhat short of the levels for the rats on the rachitic-diet-plus-KH_2PO_4 from the start of the experiment (Diet 4). The latter, similarly, gave values slightly lower than those for the group in which both KH_2PO_4 and cod-liver oil were added from the beginning (Diet 6). The analytical data relating to Group II are presented in *table 4*.

Summary. A severe rachitic diet, having a Ca:P ratio of 7.27, and resulting in good growth, produced marked reductions in the ash, Ca and P of the tibias of rats. Since the incisors grow persistently, they were expected to undergo similar changes, but this rachitic diet failed to alter the ash, Ca, and P of the teeth, as compared with animals on non-rachitic diets. When the rachitic diet was rendered non-rachitic by addition of KH_2PO_4 (2 percent), the further addition of cod-liver oil (2 percent) did not affect the ash, Ca and P of the teeth of rats ingesting it for 30 to 45 days. When this diet, as supplemented with both KH_2PO_4 and cod-liver oil, was ingested for 64–80 days, however, the Ca and P contents of the teeth were slightly greater than in animals on the diet supplemented with KH_2PO_4 alone.

References to literature. (1) Steenbock, H., and Black, A.: *Journal of Biological Chemistry*, 1925, lxiv, 263. (2) Epstein, N.: Dissertation; Columbia University, 1928. (3) Shear, M. J., and Kramer, B.: *Journal of Biological Chemistry*, 1928, lxxix, 105.

CORRELATION OF CHEMICAL AND PATHOLOGICAL CHANGES IN TEETH AND BONES ON RACHITIC AND NON-RACHITIC DIETS[1]

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In the preceding paper (1) it was noted that a severe rachitic diet, having a Ca:P ratio of 7.27, failed to reduce the ash, Ca, and P of the incisors of rats, although it produced severe reductions in these constituents of bones. The present paper deals with effects on teeth and bones of a similar diet, altered to reverse the Ca:P ratio, making it high in P and low in Ca. As in the previous study, the effect on teeth of agents curative for bone (2 percent KH_2PO_4, or 2 percent cod-liver oil) was also tested.

Experimental. The procedure was in general similar to that described in the preceding paper. As a basal diet, the previously used Steenbock and Black (2) ration (No. 2965) modified by Epstein (3), was altered by removal of the $CaCO_3$, to consist of yellow cornmeal, 66; wheat gluten, 20; egg albumin, 10; NaCl, 1; spinach, 10 grams per rat per day; water, *ad lib.* Young albino rats, 33–41 days old, ingested modifications of this basal diet as indicated in *table 1.* After 38–57 days, the animals were killed; the incisors and tibias analyzed for ash, Ca, and P, as described in the preceding paper; and celloidin sections, stained with hematoxylin and eosin, prepared from decalcified incisors and molars. Average values, and average deviations, of ash, Ca, and P are given in *table 2.* The normal values obtained for teeth (Diet 5) confirm the previous finding that the high-Ca, low-P rachitic-diet produced no effect on the ash, Ca, and P of the incisors,

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TABLE 1

Rat diets showing Ca and P contents and ratios

DIET	Ca/100 GM.	P/100 GM.	Ca:P
	mg.	*mg.*	
(1) Basal	36	170	0.211
(2) Basal + 2% KH_2PO_4	36	640	0.056
(3) Basal + 4% KH_2PO_4	36	1110	0.032
(4) Basal + 2% KH_2PO_4 + 2% cod-liver oil	36	640	0.056
(5) Basal + 3% $CaCO_3$	1236	170	7.27
(6) Basal + 3% $CaCO_3$ + 2% KH_2PO_4	1236	640	1.93

TABLE 2

Average values, and average deviations, of ash, Ca, and P for rats on experimental diets

DIET	TOOTH			BONE		
	Ash	Ca	P	Ash	Ca	P
(1) Basal	66.5 ±0.8	23.5 ±0.3	12.8 ±0.4	31.7 ±2.3	10.8 ±1.2	5.7 ±0.5
(2) Basal + 2% KH_2PO_4	65.4 ±0.6	22.8 ±0.4	12.8 ±0.2	34.7 ±4.3	12.4 ±0.9	6.5 ±0.6
(3) Basal + 4% KH_2PO_4	67.1 ±1.1	23.4 ±0.3	12.9 ±0.2	40.3 ±3.0	13.0 ±1.5	6.8 ±0.4
(4) Basal + 2% KH_2PO_4 + 2% cod-liver oil	73.0 ±1.7	25.1 ±0.5	14.4 ±0.4	51.9 ±3.3	18.2 ±0.7	9.0 ±0.4
(5) Basal + 3% $CaCO_3$	72.4 ±0.6	25.8 ±0.4	14.2 ±0.1	37.0 ±2.3	12.8 ±1.7	6.6 ±0.7
(6) Basal + 3% $CaCO_3$ + 2% KH_2PO_4	72.6 ±0.7	24.9 ±0.1	14.5 ±0.2	54.4 ±3.3	19.4 ±0.9	9.9 ±0.8

TABLE 3

Data on the relation of gain in body weight to weights of teeth and bones of rats on experimental diets

DIET	AV. WT. OF 2 INCISORS	RELATIVE WT. OF INCISORS	AV. WT. OF TIBIAS	RELATIVE WT. OF TIBIAS	AV. DAILY GAIN IN BODY WT.	RELATIVE GAIN IN WT.
	mg.		*mg.*		*gm.*	
1. Basal	77	0.65	208	0.77	0.69	0.42
2. Basal + 2% KH_2PO_4	71	0.60	201	0.74	0.53	0.32
3. Basal + 4% KH_2PO_4	69	0.58	173	0.64	0.40	0.24
4. Basal + 2% KH_2PO_4 + 2% cod-liver oil	89	0.75	230	0.85	1.30	0.79
5. Basal + 3% $CaCO_3$	88	0.74	217	0.80	0.96	0.58
6. Basal + 3% $CaCO_3$ + 2% KH_2PO_4	119	1.00	270	1.00	1.65	1.00

although the bones suffered markedly. When the Ca and P of the diet were altered, however, to make it low in Ca and either low or high in P (Diets 1, 2, and 3), a reduction of the ash, Ca, and P resulted in the incisors as well as in the tibias. The reductions in the incisors and tibias were approximately 10 percent and 30 percent, respectively. Despite the extremely low content of Ca in Diet 4 (36 mg. of Ca per 100 grams of diet), addition of cod-liver oil prevented a change in the ash, Ca, and P of incisors and tibias. When cod-liver oil (2 percent) was added to the low-Ca, low-P, and to the low-Ca, high-P diets (Diets 1, 2, and 3), after the rats had been on these diets for about two months, the ash, Ca, and P of teeth and bones rose to normal levels after two and one-half months of such feeding.

The microscopic study of incisors and molars was made with particular reference to a comparison of the chemical with the histological evidence on degrees of calcification. Most sections of incisors were made of extracted teeth, and all material was decalcified before embedding in celloidin and sectioning; hence no information about enamel was available. All the rats on the low-Ca diets (Diets 1, 2, and 3) showed changes characteristic of this type of deficiency. Although P content varied from low to high among these diets, the three groups were not distinguishable microscopically. The histological similarity of these three groups paralleled the chemical similarity. The histological evidence of poor calcification checked the chemical analyses, although the tissues seemed more severely altered than the analytical values suggested. In the incisors, calcification of the dentin was markedly poor over the length of the tooth; dentinoid occupied 40–60 percent of the total width of dentin in different regions. Various degrees of globulation in the calcified dentin were present. In the molars, broad dentinoid occurred uniformly, with only occasional irregularity of the pulp wall, and corresponding odontoblast derangement. The pulps of molars and incisors showed the cellular changes to be described in a later paper (4)—the pulps of surviving animals were either normal, or showed local or general enlargement of the stroma nuclei. In the animals that died, the stroma nuclei were shrunken, and varying degrees of vacuolation and fibrosis of the odontoblast layer occurred. Osteoid formation was marked in the alveolar bone. One animal on Diet 1 for 39 days, and one animal on

Diet 3 for 52 days, showed beginning caries in fissures of the first and second molars.

When the low-Ca Diets 1, 2 and 3 were supplemented with cod-liver oil (2 percent) after two months, and the feeding continued for two and one-half months, the alveolar bone became histologically normal, and calcification in the incisors was improved to the extent that dentinoid disappeared, but incisal dentin lining the pulp chamber became extremely irregular. The molars were also improved in these animals, but local deposits of dentinoid persisted, and varying degrees of pulp-wall irregularity and nodule formation occurred. When cod-liver oil (2 percent) was added to the basal diet plus KH_2PO_4 (2 percent) from the start (Diet 4), calcification was improved, confirming the chemical findings. The bone became histologically normal; the dental tissues, not entirely so. Moderately broad deposits of dentinoid occurred in the posterior region of the incisors, and locally in the molars, especially on the pulpal floor and in the roots. Beginning fissure-caries occurred in the first molar in one animal—on Diet 4 for 67 days. On the high-Ca diet (Diet 5), histological changes were well correlated with the chemical changes. Calcification in incisors was only regionally and slightly defective—many areas were normal or nearly so but dentin was generally somewhat globulated; other incisors showed slight dentinoid formation in the posterior part. In molars, dentinoid formation was chiefly limited to roots and pulpal floor. Alveolar bone was very poorly calcified. Beginning fissure-caries occurred in one animal in this group—on the diet for 106 days. Addition of cod-liver oil (2 percent) to this diet for two months completely restored incisors and bone histologically to normal, but local dentinoid formation persisted in the molars. Diet 6, normal in Ca and P but without cod-liver oil, produced histologically normal bone. Incisors were generally normal, although dentin was somewhat globulated, but abnormal dentinoid formation was absent. Slight local irregularity of dentin occurred in incisors. Molars were affected similarly, and presented various degrees of pulp-wall and odontoblast irregularity. Dentinoid formation was normal.

These histological changes are, it will be noted, in good general agreement with the chemical findings. The high-Ca, low-P diet, although very deleterious to bone, seems to have affected incisors only

slightly—not sufficiently for chemical detection, excepting as noted below. Molars likewise were altered notably less than on the low-Ca diets, which, whether high or low in P, produced marked changes in incisors and molars, as well as in bone. Cod-liver oil appeared, by chemical analysis, to produce normal tissues when fed to rats after a period on deficient diets or, from the start, on a diet having a markedly low Ca:P ratio. Histological examination revealed, however, that resulting improvement was incomplete. It is also interesting that an adequate Ca:P ratio without cod-liver oil produced teeth chemically normal, but showing slight histological defect. In line with this observation, the higher sensitivity of histological analysis is further indicated by the fact that changes observed histologically in incisors of rats on low-Ca diets were more marked than the chemical findings suggested; a reduction of about 10 percent in ash, Ca and P was correlated with dentin of which 40 percent or more appeared to be uncalcified. Absence of enamel in the sections might, of course, have influenced this difference.

Although chemical analyses indicated that incisors were not damaged by high-Ca Diet 5—a finding verified by histological study—the data in *table 3* show that these teeth were not exempt from general effects of this deficient diet, nor of the low-Ca diets. A comparison of relative values in *table 3*, based in each case on the value for Diet 6 as 1.00, shows that dental and osseous weight-reductions parallel roughly average gains in body weight on the various diets; and that while teeth suffered more than bones in this respect, neither was affected so markedly as body-weight values. It appears, furthermore, that relative effects of different deficient diets on tooth weight closely parallel respective effects on bones. On the low-P, high-Ca diet, the dental effect occurred as a reduction of total weight rather than an alteration in structure; while on the low-Ca diets, there was both reduction in weight and severe alteration in structure.

The occurrence of dental-fissure caries in four of these animals is an interesting finding. All these lesions, early in character, penetrated the enamel at the base of the molar fissure, and spread along the dento-enamel junction, producing the characteristic blue-staining deposit; but little or no penetration along the dentinal tubules had occurred. The distribution of these lesions among the experimental groups is

such that no etiological connection between them and the involved dietary deficiencies can be assumed. The cornmeal in these diets was about 20–30 mesh. This fact was not considered significant when these experiments were set up (in 1929), but in view of the results since obtained by the authors in another investigation soon to be described (5), it is likely that the size of the particles was the determining factor in the production of these lesions.

Summary. The effect of diets with various Ca:P ratios, with and without cod-liver oil, on the contents of ash, Ca, and P in incisors and tibias of rats was compared with the histology of their incisors and molars. The finding, previously reported (1) that a high-Ca low-P diet does not alter the teeth in the chemical aspects studied, although the bone is severely changed, was confirmed. Low-Ca diets, on the other hand, whether low or high in P, were found to produce definite alterations in the ash, Ca, and P of teeth as well as bones. Addition of cod-liver oil (2 percent), to diets otherwise deficient from the start of the experiment, or after two months of feeding and continued for two and one-half months, induced formation of chemically normal tissues. These chemical findings were in general confirmed by the histological analysis, although the latter seemed somewhat more sensitive to slight alterations. Such slight changes, in the teeth, occurred in rats on a high-Ca diet; on deficient diets plus cod-liver oil; and on a diet with an adequate Ca:P ratio but without cod-liver oil.

References to literature. (1) Karshan, M.: *Journal of Dental Research*, 1933, xiii, p. 301. (2) Steenbock, H., and Black, A.: *Journal of Biological Chemistry*, 1925, lxiv, 263. (3) Epstein, N.: Dissertation; Columbia University, 1928. (4) Rosebury, T., and Foley, G.: *Journal of Dental Research*, 1933 (accepted for publication in a succeeding issue). (5) Rosebury, T., Karshan, M., and Foley, G.: *Ibid.*

INFLUENCE OF CHLORIDES AND PHOSPHATES OF SALIVA ON ITS AMYLOLYTIC ACTIVITY[1]

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I. INTRODUCTION

In 1826 Tiedemann and Gmelin (67) noted that the stomachs of geese fed solely on a starch diet contained sugar. This observation led to the discovery by Leuchs, in 1831 (36), of amylase in the saliva. He found that after a short period of incubation with a small amount of saliva, a starch solution lost its turbidity and became sweet. He also determined that this action was specific for starch. Berzelius (4) named the enzyme "ptyalin" in 1840. Although all the salivary glands secrete amylase, the parotid secretes four times as much as any of the others, while producing almost one-half the total amount of saliva (23). Salivary amylase is present at birth (25, 38), and no report has been found claiming total absence of the enzyme in a human subject. The action of ptyalin causes the conversion of starches and dextrins to maltose (60), any glucose found in salivary digestion having its origin by other means.

Chittenden and Richards (13) found that the highest activity occurred before breakfast, and that less amylase is secreted per cubic centimeter of saliva as the glands become fatigued from excessive stimulation and secretion. During meals, and up to not more than three hours thereafter, an increase in activity occurs followed by a return to the resting level (22, 23). If the meal is omitted, the rise does not occur; if the food is chewed but not swallowed, no rise is found. Aside from these normal variations a difference in the amy-

[1] Read at the tenth general meeting of the International Association for Dental Research, Columbus, O., March 19–20, 1932; *Journal of Dental Research*, 1932, xxi, p. 436; June. The data are taken from a thesis submitted by W. Lloyd Adams to the Graduate School of Western Reserve University, June 1931, in partial fulfillment of the requirement for the degree of Master of Arts.

lolytic index of saliva from various individuals exists. The influence of psychic factors and of related reflexes complicates quantitative measurement. The mechanical action of chewing causes an increased flow of saliva; and when food is chewed, the type of secretion is determined by the nature of the food. A dry food stimulates the flow of a thin watery secretion, whereas a liquid gives rise to a more viscous saliva (51). Neilson *et al.* (48, 49) concluded that prolonged carbohydrate feeding increases the amylolytic activity of saliva. However, other workers have been unable to demonstrate any adaptation of the secretion to diet (29, 40, 41). Diseases of extra-oral nature are without effect on amylase secretion (7, 11, 46), although some workers report contrary results (18, 50). Dentifrices exert only a temporary inhibiting effect, the maximum being but twenty minutes (10, 61). Hirata (33) stated that age and sex do not influence amylolytic activity. Myers and Dellenbaugh (46) were unable to demonstrate any racial difference.

Numerous methods have been advanced for the determination of the amylolytic activity of the saliva. These methods may be classed as either (1) the "liquefaction" or "iodine" methods (52), whereby the disappearance of starch in the digestion mixture is determined with the starch-iodide blue color as an indicator; or (2) the "saccharification" methods, whereby the products of hydrolysis are measured. Both methods are based upon what has been called Kjeldahl's "law of proportionality" (35), which states that as long as the substrate is in considerable excess, a direct proportion exists between the amount of amylase present and the amount of starch hydrolyzed. The iodine method was used in 1881 by Roberts (56). In 1908 the technique was put upon a more exact basis by Wohlgemuth (70). Sherman, Kendall and Clark (63) called attention, in 1910, to certain errors in this method. Evans (24), studying the methods of Roberts and of Wohlgemuth, defined their limitations. Measurement of the digestion products has been accomplished in various ways. Kjeldahl (35), Lintner (37), Chittenden and Ely (12), etc. (9, 27), depended upon the reducing action of the sugar on an alkaline copper-reagent, usually Fehling's solution. The resulting cuprous oxide was measured gravimetrically by most workers until 1910 (63). Bang (2) used a titration method to determine the reduction. Colorimetric methods

then came into use. Myers and Dellenbaugh (46) applied the picric-acid reduction method, and later other workers employed colorimetric copper-reduction methods. Recently Myers and Reid (47) have reinvestigated these two procedures, and regard picric acid as definitely superior to copper owing to the fact that it yields nearly twice as much reduction with maltose.

A suitable method for amylase estimation must recognize the various factors that affect the action of the enzyme. The substrate most commonly used is potato-starch solution, since the readiness with which it is hydrolyzed is exceeded only by a solution of arrowroot starch (55). In certain methods the fact that the degree of dispersion of the starch greatly influences the rate of hydrolysis (54) should be considered. The temperature optimum for ptyalin action was found by Roberts (56) to be from 35° to 40°C. Blake (6) stated that at 46°, heat begins to inactivate the saliva. It is interesting to note that maltose, glucose, Cl^-, Br^-, or serum, protect dialyzed saliva to a certain extent from heat inactivation (34). The ions NO_3^- and I^- do not protect. A $\frac{1}{10}$ *N* solution of HCl completely destroys amylase; an excess of alkali does so with equal readiness. The optimum pH for salivary-amylase activity is placed at pH 7 or slightly lower (3, 53, 62). Chittenden and Smith pointed out that in determining the amount of products formed by a method of starch hydrolysis, the relation of amount of hydrolysis to amount of enzyme is more directly proportional when the saliva is diluted 50 to 100 times. With their method Myers and Dellenbaugh (46) found that it was necessary to carry out amylolysis on saliva diluted 200 times in order to secure a direct proportionality of amylolytic action: 0.01 cc. of saliva was allowed to act upon 10 mg. of soluble starch in a volume of 2 cc.

One of the most important factors affecting amylolytic digestion is the electrolyte content of the digestion mixture (16). Dialysis inactivates ptyalin (2, 30, 44). Of the various salts that reactivate ptyalin, it has been found that in most cases the anions have far greater influence than the cations (2, 17, 57, 58); of the former, the chloride ion exerts the strongest influence. Fricker (28) found that NaBr may replace NaCl with practically the same activation. Excess of NaCl causes no diminution of activity until 13 percent is present.

Rogers (59) stated that the phosphate ion was necessary for amy-

lase action, but Prinz (53) obtained good activity with dialyzed saliva to which only NaCl had been added. Nevertheless, the phosphate ion exerts an influence on ptyalin. Bang (2) found that mild activation resulted with small quantities of mono-sodium phosphate. Broeze (8) has studied the influence of certain electrolytes on dialyzed saliva. Dryerre (20) proved that thiocyanate acts neither as a stimulator nor inhibitor of ptyalin action. Since chlorides and phosphates constitute the chief electrolytes of saliva, they should, in view of their

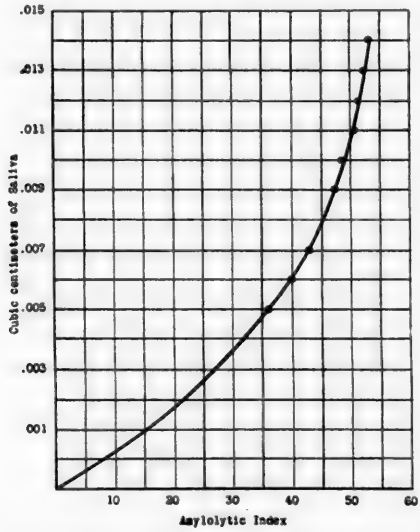

FIG. 1. CURVE OBTAINED BY PLOTTING AMYLOLYTIC INDICES AGAINST RELATIVE AMOUNTS OF AMYLASE USED TO OBTAIN THE INDICES

activating property, exert some influence upon the activity of ptyalin. No correlated study of amylolytic activity and the salt concentrations of the saliva having been found in the literature, this investigation was undertaken.

II. METHODS EMPLOYED

The Myers and Dellenbaugh (46) method for the estimation of *amylase content* was used. One cc. of saliva is diluted with distilled water to 100 cc., and 1 cc. of the diluted sample is incubated at 40°C. for exactly 30 minutes with 1 cc. of 1 percent soluble-potato-starch solution (total dilu-

tion, 1–200). The resulting reducing products are measured colorimetrically by the picric-acid method, as described by Myers and Bailey (45). In order to study the effect of increased NaCl concentration, each sample was also analyzed after making the 1:100 dilution with a 0.3 percent NaCl solution. The percentage of starch hydrolyzed gives the amylolytic index of the saliva. To determine whether the indices so obtained are a true estimate of the amount of amylase present, the following experiment was carried out. A uniform sample of saliva was diluted 1:50, and from this other dilutions were made so that 1 cc. contained absolute amounts of saliva ranging from 0.005 cc. to 0.013 cc. These amounts were plotted as ordinates, and the corresponding amylolytic indices found as abscissae. The observations are recorded in *fig. 1.* The range of indices encountered is from about 20 to 55, with a majority between 30 and 45; so that, for purposes of comparison, the amylolytic index is practically a true estimation of the amount of amylase present, since this section of the curve approximates a straight line. For *chlorides* the Van Slyke method (68) and the Volhard titration were used. Owing to the small amount of protein in saliva, it was unnecessary to digest the samples as is done in the Wilson and Ball technique (69). Several writers have shown that saliva contains only a very small amount of organic phosphorus (64, 71). Therefore the *total phosphorus* is a comparable measure of the amount of phosphate influencing ptyalin activity. The Fiske and Subbarow method (26) for phosphorus estimation, slightly modified, was employed. Two cc. of saliva are digested by a sulfuric-nitric acid mixture (1:2) to destroy protein. The diluted residue is determined as usual. It is essential that the amount of acid in the unknown and in the standard (0.3 mg. of P) be the same.

III. EXPERIMENTAL

The subjects of this study were medical and dental students, selected because of comparable rates of saliva secretion. Each collected his saliva, without artificial stimulation, by allowing the spontaneous flow to filter through a small filter paper into a clean test tube. This method seemed best because a review of the literature (13, 14, 15, 53, 65) indicated certain differences in saliva stimulated by artificial means. The collection was made during morning laboratory periods, two to three hours after breakfast. It was observed that the index of an individual did not vary to any extent in health. To study this more closely, special attention was given to the collection of saliva from two members of the staff. With students who occasionally omit breakfast, and otherwise vary the normal conditions, the degree of uniformity under which the samples could be obtained would

TABLE 1

Constancy of amylolytic index in health

YEAR.....	1930			1931							
DATE.....	12/15	12/16	12/17	1/7	3/13	3/16	3/20	3/27	4/20	4/21	4/22
SUBJECTS											
1	40	37	51	41	40	41	39	41	39	40	41
2	33	33	33	32	37	33	35	35	31	33	33

TABLE 2

Amylolytic index in relation to chloride and phosphate contents of saliva, and condition of teeth and gums

SUBJECT	AMYLOLYTIC INDEX			CONTENT IN SALIVA		CONDITION OF TEETH AND GUMS
	Water dil.	NaCl 0.3% dil.	Difference	NaCl Mg. in 100 cc.	Total P Mg. in 100 cc.	
16	33	55	22	58	13	Good
2	35	55	20	57	15	"
20	32	49	17	78	18	"
10	25	41	16	65	11	"
Average first four.....	31	50	19	65	14	
23	41	57	16	68	17	"
15	42	58	16	81	14	"
7	35	50	15	89	17	Fair
5	46	60	14	86	18	Poor
25	40	54	14	58	15	Good
26	37	50	13	101	17	"
21	40	53	13	102	14	"
1	40	53	13	75	14	"
18	43	55	12	78	18	"
8	42	53	11	90	16	"
24	47	58	11	103	16	Recent ext.
22	45	56	11	115	18	Good
9	42	51	9	107	13	"
4	46	55	9	115	16	"
3	40	47	7	107	22	"
6	48	55	7	111	20	Poor
14	44	50	6	132	21	"
Average last four.....	45	52	7	116	20	

scarcely be equal to that in these two cases. As is noted in *table 1*, the amylolytic index remained constant over a period of five months.

To show the influence of the chloride and phosphate content of the saliva on the amylolytic index, a study was made on 21 subjects (*table 2*). Each figure assigned in that table to an individual represents the average of five determinations on different days over a five-week period. The results

TABLE 3

Influence of severe colds on amylolytic index

Determinations made on days of greatest severity are indicated by italic.

SUBJECT	AMYLOLYTIC INDEX		CONTENT IN SALIVA		REMARKS
	Water dil.	NaCl 0.3% dil.	NaCl Mg. to 100 cc.	Total P Mg. to 100 cc.	
11	43	57	118	15	Normal
	46	42	107	18	"
	21	33	83	15	Severe coryza
	18	—	118	16	" "
	29	—	105	15	Recovering coryza
12	27	47	83	10	Slight coryza
	25	60	118	12	" "
	24	*40*	89	11	Moderate coryza
	29	—	107	12	" "
	53	—	83	11	Recovery
13	34	41	72	10	Normal
	36	57	79	19	"
	26	31	66	11	Slight coryza
	18	—	60	12	Severe coryza
	34	—	72	10	Recovery
17	*22*	*35*	108	—	Severe coryza
	41	53	119	—	Recovery
	32	46	96	12	Normal
	32	52	90	12	"
19	35	37	154	—	Normal
	31	46	109	—	Slight coryza
	19	*48*	127	14	Severe coryza
	36	53	94	16	Normal

arranged on the basis of increased activity brought about by the NaCl diluent disclose a corresponding difference in salt concentration of the saliva. Averaging the figure for the four subjects at the top, we find that, with an average increase in the index of 19 (a rise from 31 to 50) when 0.3 percent NaCl is used as the diluent, the NaCl content of the saliva averaged 65 mg.

per 100 cc.; the total P, 14 mg. But with the four individuals at the bottom of the list, showing an average increase in the index of 7 (a rise from 45 to 52), the NaCl content of the saliva averaged 116 mg.; the P, 20 mg.

Four of the twenty-six students suffered severe colds in the course of the investigation. Simultaneously, it was surprising to find considerable reduction in the amylolytic index, with the lowest index appearing during the most distressing period of the cold. *Table 3* shows the results; the determination made at the height of the cold is indicated with italic.

IV. DISCUSSION

Table 1 shows that, with one exception, there is a very marked constancy in a given individual's amylolytic index over a period of five months. There was a maximum difference of 4 in the index of Subject 1 throughout the period, and a difference of 5 in the case of Subject 2. Considering the error of the method, it is very probable that an individual's amylolytic index is even more constant than that shown by these figures.

The chloride and phosphorus contents exerted very definite effects, an average of 31 in the index accompanying an average chloride content of 65 mg. and P content of 14 mg., while an average index of 45 accompanies an average chloride content of 116 mg. and P content of 20 mg. The further evidence—that addition of NaCl to the saliva raised the amylolytic activity more in the cases showing low chloride contents—supports the conclusion that the chloride and phosphate contents exert important influences on the amylolytic activity of saliva.

Since factors affecting the amylolytic index were being sought, the conditions of the teeth and gums of the subjects were recorded. For this examination we are indebted to the University Health Service. From *table 1* it may be seen that the four individuals with the lowest indices—25, 32, 33, 35—had good teeth and gums. On the other hand, the three individuals whose rating was poor, together with the one who had had a recent extraction, all showed the highest indices—44, 46, 47, 48. These findings suggest that a high amylolytic index may be a factor in the development of dental caries, provided acid fermentation of carbohydrate (maltose) is an etiological factor in caries. Obviously, much more work is needed before a definite conclusion can be drawn on this important question.

Explanation for the lowering of the index in severe colds is lacking. It did not appear to be due to a change in the rate of secretion. It is suggested that the continuous irritation from coughing may call for a more steady secretion, and that overwork of the salivary glands may lead to a lower amylase secretion than normal. With relief from coughing, the secreting cells would then have more time to synthesize a normal amylase content, thus accounting for the rapid return to normal that was manifested in recovery from the cold.

V. SUMMARY

A correlated study of amylolytic activity, chloride content and phosphate content, of saliva was carried out on twenty-four medical and dental students, and on two members of the staff.

The normal range of amylolytic index is 25 to 50. However, the index of a normal individual is unusually constant. In one individual the index varied but $\pm$ 2 points over a period of five months.

The variation of amylolytic index among individuals is shown to be closely related to the chloride and phosphorus contents of the saliva. On the average, the lower amylolytic indices accompany the lower chloride and phosphorus contents; the higher indices are accompanied by the higher chloride and phosphorus contents. An average of 45 percent increase in index occurs with a chloride increase of 79 percent and a P increase of 43 per cent.

It is suggested that a high amylolytic index may have an effect on the condition of the teeth and gums, and that this in turn may be an etiological factor in dental caries, provided acid fermentation of carbohydrate is a cause of tooth decay.

During severe colds the amylolytic index is definitely lowered by as much as 50 percent. It is suggested that this be may due to fatigue of the salivery glands accompanying a constant flow occasioned by the irritation of coughing.

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AN ATTEMPT TO DISSOCIATE CERTAIN ORAL AND DENTAL STRAINS OF LACTOBACILLI, AND SOME NOTES ON DIFFERENTIATION OF THE ROUGH VARIANTS FROM INTESTINAL LACTOBACILLI[1]

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Various investigators have ascribed a definite rôle to lactobacilli in the production of dental caries, and in some instances have referred to the lactobacillli as *L. acidophilus*. In support of the use of this name certain observations are cited in which rough variants were derived from the characteristically smooth or l types. Complete evidence has not as yet been forthcoming that these rough variants acquired the cultural and physiological properties possessed by the bona fide intestinal type which they resemble more or less. Goadby (1910), Baumgartner (1913), Hartzell and Henrici (1917), Howe and Hatch (1917), Seitz (1921), Rosebury, Linton and Buchbinder (1929), Hadley, Bunting, and Delves (1930), and Hewitt and van Meter (1930), constitute the main group of investigators who have claimed a definite etiological relationship of *Lactobacillus* genus to dental caries, and have assumed these dental strains or types to be identical with *L. acidophilus* of intestinal origin. McIntosh, James, and Lazarus-Barlow (1924) recovered the same (dental) type of organism from various mouths but, after further studying its characteristics, concluded that it differed in some respects from the intestinal lactobacilli. Years of experience of this laboratory with intestinal aciduric organisms, and with those of dental and oral origin, have convinced the writers that the intestinal and oral (including dental) types of lactobacilli are separate and distinct forms. More recent

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studies have tended to further confirm their views. In a recent publication (1932), Rettger makes the following statement:

"The dental type which is found [according to some investigators[2]] in such large numbers in tooth cavities and in the saliva of carious mouths has certain characteristics which should distinguish it from the generally recognized intestinal *L. acidophilus*. It produces, as a rule, smooth, solid, comparatively large and dense colonies on appropriate agar plates, when grown at incubator temperature. It grows readily at room temperature. Its cultural requirements are not extremely exacting. It is a rapid and high acid-producing organism and acts in a wider carbohydrate range than does *L. acidophilus*. Finally, there is no evidence to show than an aciduric organism of the odontolyticus type occurs in the intestine ordinarily or following a high lactose or dextrin diet."

The present work was undertaken to determine to what extent dental and oral strains of lactobacilli can be converted into rough (colonial) variants that resemble the intestinal "x" types, and maintained as such; and also to determine whether such variants acquire the cultural and physiological characteristics of recognized intestinal types.

Strains employed in this work and their colonial forms: A. Oral and dental strains.—L-42 and L-43 were obtained from Rodriguez. 33 was from the Morishita collection. S-5, 13, and 19—all producing small, densely granular colonies with more or less even, entire border—were from the Morishita collection. R-63 and R-67 were received from Rosebury. *B. Intestinal strains.*—Scavano (human): *L. acidophilus*, this laboratory; small delicate, extremely fuzzy "x"-type colonies. R-1 (rat) and R-3 (rat): *L. acidophilus*, this laboratory; "x" type, but less delicate than the Scavano strain.

Method of dissociation. The medium used to promote dissociation was the tomato broth of Kulp (1927). When a solid medium was required agar (2 percent) was added to the liquid medium. The inoculated tomato-broth tubes were incubated at 37°C. for 48 hours. Some of the culture material was then streaked on tomato agar and incubated at 37°C. for 48 hours. Transfers were made to tomato broth from the smoothest and roughest colonies. After 48-hour in-

[2] Inserted by the present authors.

cubation at 37°C., agar plates were again streaked, and the process of selection and isolation repeated for a period of approximately four months, with the constant aim of developing extreme smooth and rough variants. In several instances 0.5 percent lithium chloride was added to the tomato broth with the hope of hastening the dissociation. The use of the lithium chloride was discontinued when it was found to confer no special benefit. Poured tomato-agar plates were prepared at bi-weekly intervals, to study colonial morphology of the variants in deep agar, as well as on the surface.

RESULTS OF THE DISSOCIATION ATTEMPTS

The so-called normal colonial morphology of the oral and dental strains is illustrated in *fig. 1* (Strain 33) and *fig. 2* (Strain 19), which fairly represent the entire group. The only organism in the list of oral and dental strains from which a distinctly rough variant was secured by the above procedure was No. 19. The colonial morphology of this variant is shown in *fig. 3* (surface colony) and in *fig. 4* (deep colony). An intermediate or RS type was also secured from this strain (*fig. 5*). It was impossible, however, to maintain the rough variant for more than three transfers. After this, smooth colonies were always seen on the plates along with the definitely rough. Attempts to preserve the distinctly rough-colony type were abandoned after many repetitions. None of the other mouth and tooth strains employed could be converted into a definitely rough type. The nearest approach to a rough variant was the RS type illustrated in *fig. 6*; this moderately rough variant constantly tended to revert to SR and S types.

No attempt was made in the present study to derive S and SR variants of the *Scav.* intestinal type of *Lactobacillus*, which has long been accepted as a typical and authentic human strain of *L. acidophilus*. Rettger and Gillespie (unpublished work) were unable, after long and continued efforts, to obtain smooth or even semi-smooth variants, except transitory forms which did not retain their new colonial characteristics long enough to permit determination of their general cultural and physiological properties. The smooth variants always reverted quickly to the RS and original R types.

Morphology of colonies in deep agar (*poured plates*). At the begin-

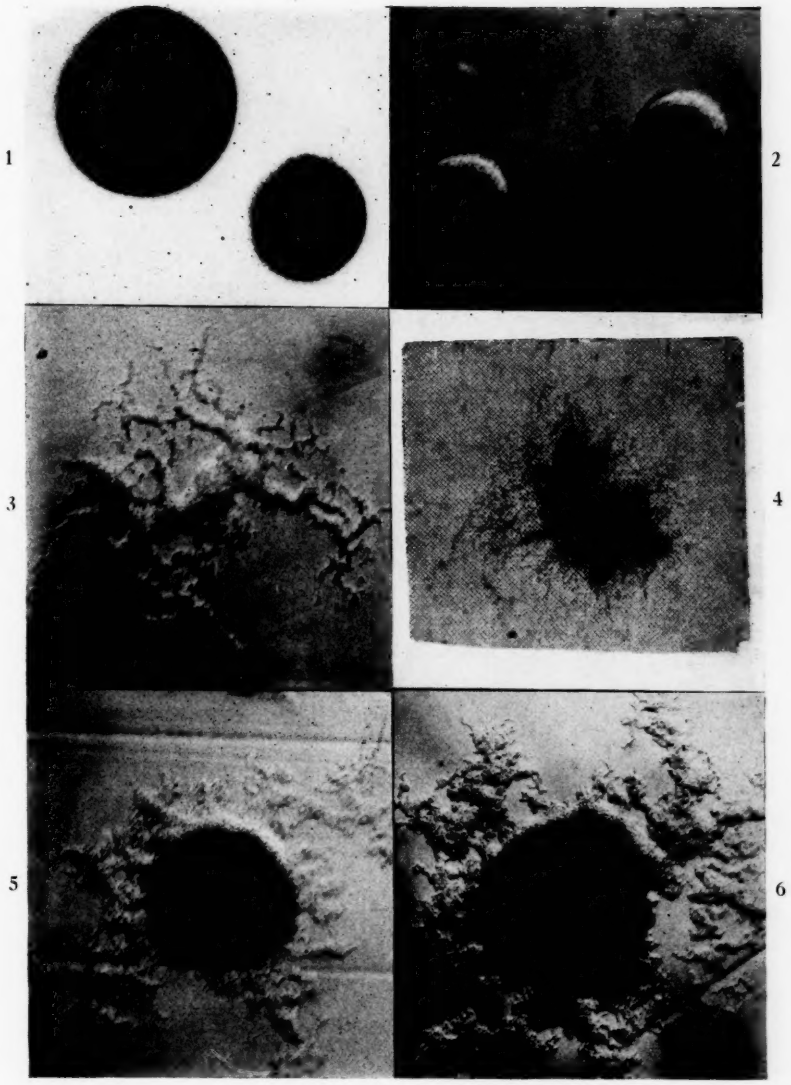
ning of this work, all the strains excepting No. 19 and the intestinal types produced solid, dense, deep colonies with smooth borders. When the efforts to derive rough variants were finally abandoned, the colonial morphology of these different strains had at best changed only to the extent that the colonies appeared more granular than at the outset, with the edges only moderately irregular and at times very slightly fuzzy, even in crowded plates. Crowding of plates was avoided, as a rule, because of the tendency that some so-called smooth strains have to assume a more or less rough character when the plates are heavily seeded. Strain No. 19, when the most extreme dissociation was effected, produced deep colonies in poured plates, which to a very large extent resembled those of the normal *Scav.* intestinal strain. This variant type is illustrated in *fig. 4.* When the surface colonies on the streaked plates were of the RS form, the deep colonies of the same variant in poured plates were also of the RS type, and resembled the transitory RS forms obtained at times from the other oral and dental strains studied.

Growth on sugar-free medium. At the beginning and the conclusion of these experiments all oral and dental strains of lactobacilli grew in yeast broth containing no demonstrable carbohydrate, irrespective of whether the S or the temporary RS or R types or variants were employed. The growths were not as luxuriant, however, as in the sugar-containing medium. In contrast to these observations, the three strains of *L. acidophilus* failed to produce colonies that were at any time visible to the unaided eye.

Growth in peptone water. Each of the oral and dental strains grew well in peptone water, with or without added carbohydrate, no appreciable differences between the S, RS, and R types having been observed. On the other hand, *L. acidophilus* strains never produced any visible growth in this medium.

Growth at ordinary room temperature. All strains of oral and dental origin grew well at 20–22° and 37°C. This was equally true of the S, RS, and R (No. 19) types. The three aciduric strains of intestinal origin at no time produced visible growth at 20–22°C., but readily developed characteristic macroscopic colonies at 37°C.

The observation on growth in carbohydrate-free media, and at ordinary room temperature (20–22°C.), are in harmony with previous experiences of this laboratory.



FIGS. 1 AND 2. COLONIES OF NORMAL OR UNDISSOCIATED DENTAL LACTOBACILLUS; STRAINS 33 AND 19, RESPECTIVELY

FIG. 3. SURFACE COLONY OF ROUGH VARIANT OF DENTAL STRAIN NO. 19

FIG. 4. DEEP COLONY OF ROUGH VARIANT OF DENTAL STRAIN NO. 19

FIG. 5. INTERMEDIATE, OR RS TYPE, COLONY OF DENTAL STRAIN NO. 19 ON SURFACE AGAR

FIG. 6. RS TYPE OF VARIANT OF DENTAL STRAIN NO. 33 IN SURFACE AGAR

DISCUSSION AND CONCLUSIONS

No attempt is made here to present a comprehensive report on dissociation, and on the interrelationships, of the oral and intestinal types of aciduric organisms. The main aim is to call attention to certain differences between recognized oral (including dental) strains and those of known intestinal origin, which do not disappear, according to the present study, in the process of dissociation of oral strains into the RS or R types.

Complete dissociation of oral and dental strains into the R form was accomplished with one organism only: No. 19. The R colonies of this strain closely resembled the typical colonies of *L. acidophilus*, but tended constantly to revert to the less rough and the smooth types.

In the RS and R variants or dissociants of the dental strain, No. 19, the original ability to grow in peptone broth and in carbohydrate-free media, and to develop macroscopic colonies at low (20–22°C.) temperature, was preserved. These properties are in marked contrast to those of *L. acidophilus* of strictly intestinal origin. Differences between the oral and dental types are not limited, however, to these criteria, as the authors will attempt to show in future publications.

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